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THE RELATION OF SUBACUTE AND ACUTE BACTERIAL ENDOCARDITIS TO RHEUMATIC ENDOCARDITIS*

A Study of 66 Cases With Necropsies

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THE studies of Shotmüller, Libman, Thayer, Horner, and others have made the clinical recognition of subacute and acute bacterial endocarditis relatively simple. In the past, the diagnostic criteria of the disease, variations in the clinical course, and the morphology and bacteriology of the cardiac lesions have been the center of interest. The relation of subacute bacterial endocarditis to rheumatic endocarditis, congenital deformities of the valves, and to other structures of the heart and of adjacent blood vessels, has also been stressed. At the present time, however, an exact description of certain aspects of the development and natural history of subacute and acute bacterial endocarditis in clinical medicine is lacking.

This report summarizes certain clinical and morphological relationships between subacute bacterial endocarditis and rheumatic heart disease. The data used were taken from a series of 5215 consecutive necropsy records covering a period from 1905 to 1929, and compiled under the direction of Dr. Frank B. Mallory at the Boston City Hospital. The necropsy records of the bacterial endocarditis cases, together with clinical data, were analyzed for age distribution at death and for the distribution and frequency of an underlying rheumatic valvulitis. This information could not be satisfactorily obtained from cases selected primarily from clinical material, as the latter usually includes only the more typical cases and leaves the less clearly defined ones unrecognized.

There were 47 cases of subacute bacterial endocarditis, 35 of which were superimposed definitely on an old rheumatic lesion. The remaining 12 gave insufficient gross morphological evidence of an underlying rheumatic valvulitis, although in six, history of rheumatic fever was obtained. Nineteen additional cases were classified as acute bacterial endocarditis, there being evidence of either a rheumatic or a congenital basis in every case.

As there are no universally accepted diagnostic criteria of subacute bacterial endocarditis, the separation of the cases of subacute from those of acute bacterial endocarditis was difficult in some instances. A majority of investigators restrict the use of subacute bacterial endocarditis to an endocarditis caused by the streptococcus viridans and usually with vegetations containing large numbers of these organisms. The disease, as generally defined, runs a subacute course of months in duration, produces septic emboli and usually shows little or no ulceration at the site of the vegetation. The gonococcus, the meningococcus, and other organisms may also produce an endocarditis with a subacute course, and thus in some classifications, subacute bacterial endocarditis also includes cases caused by these organisms.

Acute bacterial endocarditis, on the other hand, is generally caused by the staphylococcus aureus or streptococcus hemolyticus, less commonly by the pneumococcus and other organisms. These bacteria induce a more acute picture with rapid course, frequent chills, a high, picket-fence type of temperature record, pyemic infarcts, and destruction or perforation of the valves at the site of the vegetative process.

In the late stage of subacute bacterial endocarditis, the blood culture occasionally yields streptococcus hemolyticus or staphylococcus aureus although previously, in the same patient, the culture had been either negative or positive for streptococcus viridans. The vegetations in such cases often show a process that is partially acute, with some resulting destruction. Whether streptococcus viridans, through a change between the interrelation of the microbe and host, can ever change to streptococcus hemolyticus is an open problem. It is more probable that in these cases more virulent bacteria secondarily invade the blood stream and cause an acute terminal pyemic process. This apparently occurred in a few of the cases listed as subacute bacterial endocarditis. All hearts showing a vegetative process caused by the streptococcus viridans, as discovered by antemortem or by postmortem heart cultures obtained soon after death, were listed in the subacute group, since the other findings, with the exception of a very short duration of the clini-

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cally recognized illness in a few cases, were consistent with the clinical picture of this disease. The deaths following such a short illness were generally due to acute complications caused by emboli.

BACTERIOLOGY

One or more blood cultures were taken during life in 22 of the 47 subacute cases, chiefly those admitted in the past fifteen years. In 14, the cultures were positive; 11 showed streptococcus viridans; one, streptococcus hemolyticus; one, staphylococcus aureus; and one, a strepto-diplococcus resembling the organism of Poynton and Paine. The clinical course of the case in which the streptococcus hemolyticus was discovered resembled that of subacute bacterial endocarditis. In two cases in which antemortem blood cultures were not available, blood taken from the heart on postmortem examination contained streptococcus viridans. In three other cases organisms were cultured from the vegetations; streptococcus viridans in one, and streptococci of undetermined type in the other two.

Staphylococcus aureus was isolated from the vegetations, from blood obtained from the heart, or from antemortem blood cultures in eight of the 19 cases of acute bacterial endocarditis. Streptococci of undetermined type were isolated in five cases, gram negative diplococci in two, and a mixed infection with staphylococci, streptococci, and pneumococci in another. In three cases unidentified cocci were found in smears from valves and vegetations.

DURATION OF ILLNESS AND HISTORY OF RHEUMATIC FEVER

Table I indicates the duration of the illness in subacute bacterial endocarditis from the date of the recognition of symptoms. Twenty-seven, or 64 per cent of the cases, lasted from two to nine months. In eight, or 19 per cent of the cases, the disease existed one year or longer. The shorter duration of the disease is not necessarily due to lack of recognition of symptoms on the part of the patient or of the physician. Thus, in the case of a medical student who for years suffered from rheumatic heart disease and feared subacute bacterial endocarditis, the disease lasted but four months after the discovery of the first petechia by the patient. This petechia was recognized after practically daily search for evidence of subacute bacterial endocarditis during the three years preceding.

TABLE I

DURATION OF CLINICAL COURSE OF SUBACUTE BACTERIAL ENDOCARDITIS FROM TIME OF RECOGNITION OF SYMPTOMS IN 42 CASES

Duration	Number of Cases	Percentage of Total Cases
1 week or less	3	7.2
2 weeks	1	2.4
1 month	2	4.7
2 months	6	14.3
3 months	5	11.9
6 months	14	33.3
9 months	3	7.2
1 year or over	8	19.2

Rheumatic fever was definitely mentioned in the clinical history as present or absent in 33 of the 47 cases. Rheumatic fever had occurred in 22 of these 33 cases.

INCIDENCE, AGE AND SEX DISTRIBUTION

The occurrence of 47 subacute and 19 acute cases of bacterial endocarditis among 5215 necropsies gives an incidence of 0.9 per cent and 0.4 per cent respectively. As there were 474 cases with evidence of rheumatic heart disease in the entire series, it may be said that subacute bacterial endocarditis occurs about once to every 10 cases of rheumatic heart disease, and acute bacterial endocarditis approximately once to every 25.

There were 164 deaths caused by rheumatic heart disease in the entire necropsy series. The ratio of the total cases of bacterial endocarditis to fatal rheumatic heart disease is thus 66 to 164; and the ratio of acute and of subacute cases, respectively, to fatal rheumatic heart disease, 19 to 164, and 47 to 164. Thus, for every four deaths due to fatal rheumatic heart disease there is one from subacute bacterial endocarditis.

The age distribution at death in the subacute cases is indicated in Chart 1. Twenty-five of the 47 deaths from subacute bacterial endocarditis occurred after the age of 40; seven after the age of 50 years. The highest frequency is observed in the fourth and fifth decades. Three deaths occurred after the age of 60; none before the age of 15 years. The age distribution at death from subacute bacterial endocarditis does not correspond to the age distribution at onset of rheumatic fever. Rheumatic fever before the age of 15 years is common, while subacute bacterial endocarditis is rare. Seventy-five per cent of a group of patients dying from rheumatic heart disease had their first infection before the third decade.

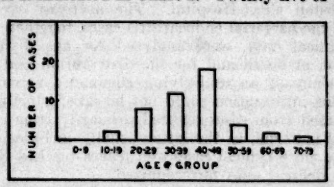

CHART 1. Age Distribution at Death of 47 Cases of Subacute Bacterial Endocarditis.

The age at death distribution curve for 467 cases of rheumatic heart disease indicates a preponderance occurring in the fourth and fifth decades (Chart 2). A comparison for subacute

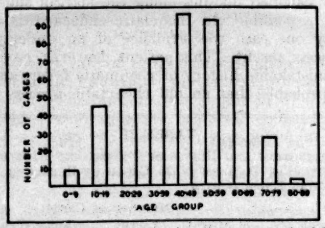

CHART 2. Age Distribution at Death of 467 Cases of Rheumatic Heart Disease.

bacterial endocarditis shows that the maximal number of deaths occurs somewhat earlier in subacute bacterial endocarditis. After the fifth decade there is a rapid fall in the frequency distribution curve of subacute bacterial endocarditis, while the frequency curve for death due to rheumatic heart disease falls relatively slowly. The low incidence of subacute bacterial endocarditis in the first and second decades may also be related to the greater prevalence of activity in these decades. In the group of subacute cases only one case was found in which apparently an acute rheumatic endocarditis was associated with the subacute process. Active rheumatic endocarditis was present in approximately 80 per cent of patients with rheumatic heart disease who died before the age of 20, but in only 25 per cent of patients dying after the age of 50, in our analysis[1].

Chart 3 shows the age distribution of 18 pa-

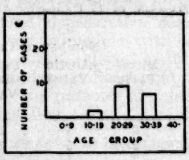

CHART 3. Age Distribution at Death of 18 Cases of Primary Acute Bacterial Endocarditis.

tients who died from acute bacterial endocarditis. All the deaths occurred below the age of 40.

In the present series, cases of subacute and acute bacterial endocarditis were more common in the male in contrast to rheumatic fever, which appears to have a somewhat greater incidence among females[2]. Of the subacute cases 34 were in males, and 13 in females; of the acute cases 14 were in males, and 5 in females. The ratio of male to female is thus approximately 3 to 1. Blumer[3], and Fulton and Levine[4] also noted the higher incidence of subacute bacterial endocarditis among males. This lack of parallelism between the sex distribution of subacute bacterial endocarditis and rheumatic heart disease further suggests that bacterial endocarditis is not etiologically related to rheumatic endocarditis except secondarily, in so far as the latter furnishes a valvular injury which serves as an area of low resistance for the growth of bacteria.

RELATION OF CHRONIC VALVULAR DISEASE TO DEVELOPMENT OF SUBACUTE BACTERIAL ENDOCARDITIS

Subacute bacterial endocarditis generally occurs as a vegetative process engrafted on the site of an old rheumatic endocarditis. Occasionally a congenital and possibly in rare instances a syphilitic lesion is the site for its development. Some authors believe that there is always disease or injury, however slight, that serves as the ground for the implantation of the bacteria, and that the condition never occurs as a primary process on a normal valve. The difficulty of identifying a mild rheumatic process in the presence of vegetations with bacterial growth, which not infrequently cause some destruction, makes this assertion difficult to prove. In 12 cases in our series, for example, gross examination of the valves revealed no evidence of an old underlying valvular process, although six of the 12 cases had histories of rheumatic fever. Such specimens were often preserved in gross, and no histological examination was made. An underlying process may, of course, have been present, but obscured by the overlying vegetative growth. In three cases, the hearts were examined in great detail by us, but neither gross nor microscopic examination revealed evidence of previous chronic injury.

The chronic valvular lesions due to rheumatic fever were graded according to the degree of damage in 474 cases in which any degree of rheumatic heart disease was present at death. Grade I included the earliest changes, consisting in definite thickening of the free margin of the valves; Grade II included cases in which there was further destruction described as scarring, puckering, retraction, moderate thickening, and large vegetations on the free margin but without stenosis of the orifice; Grade III included only cases that showed some degree of stenosis; Grade IV included hearts with marked valvular stenosis, the ring admitting a finger-tip at most, and with the mitral orifice usually described as "fish-mouth" or "button-hole". The 35 cases with subacute bacterial endocarditis showing definite gross morphological evidence of underlying rheumatic disease are so classified in Table II.

In the group of 474 rheumatic cases the mitral lesions were classified in Grade III in 85 cases and in Grade IV in 57 cases, giving a total of

142 with stenosis. Of the 35 cases of subacute bacterial endocarditis with rheumatic lesions, eight were classified as Grade III and four as Grade IV, making a total of 12 with stenosis. The ratio of cases with stenosis (Grades III and IV) to the entire rheumatic group is 142 to 476, which is comparable to the ratio of the rheumatic distribution among the total subacute cases: 12 to 35, or 12 to 47 if one believes that all subacute cases have a rheumatic basis.

Fulton and Levine[4] have stated that mitral stenosis is uncommon in the presence of subacute bacterial endocarditis and they attributed this to some form of incompatibility between mitral stenosis and subacute bacterial endocarditis, stating that hearts with advanced stenosis are less likely to develop this complication.

The statistical analysis of morphological findings noted above, however, would rather indicate that any degree of rheumatic endocarditis, however slight, is equally liable to favor the growth of bacteria and to be the basis of subacute process; that mitral stenosis occurs as frequently in subacute bacterial disease as it does in a group of unselected rheumatic heart cases; and that owing to the relatively larger number of rheumatic hearts with very mild lesions the subacute cases with marked mitral stenosis are correspondingly infrequent.

The cardiac rhythm was determined in 34 cases of subacute bacterial endocarditis and was found to be regular in 32 and irregular (auricular fibrillation) in two. The rarity of decompensation and auricular fibrillation in subacute bacterial endocarditis (Libman[5] and Fulton and Levine[4]), in our opinion, may be explained on the same basis as the apparent infrequency of mitral stenosis. Auricular fibrillation is particularly associated with advanced type of mitral stenosis and dilated auricles in rheumatic heart disease. As in subacute bacterial endocarditis, there exist considerably more cases with mild than with severe rheumatic valvulitis and since, according to our belief, a slight lesion is just as liable to give rise to the disease as is a marked one, it is to be expected that the vast majority of cases will be unassociated with decompensation and auricular fibrillation.

VALVULAR DISTRIBUTION OF OLD RHEUMATIC LESIONS AND BACTERIAL VEGETATIONS IN SUBACUTE AND ACUTE BACTERIAL ENDOCARDITIS

The distribution of the chronic rheumatic valvular lesions in 35 of the 47 cases of subacute bacterial endocarditis is shown in Table II. As has been mentioned, there was no gross morphological indication of an underlying process in 12 cases. If a rheumatic basis had been present in these cases, they would have been classified as Grade I.

Table III shows the location of the rheumatic lesions in 17 of the 19 cases of acute bacterial endocarditis. A congenital bicuspid aortic valve was the basis in an additional case which showed, besides the destructive process in the heart, a few calcified nodules along the margin suggesting a possible old rheumatic endocarditis. In only one case was evidence of an underlying process absent. This patient, however, gave an unmistakable history of rheumatic fever, and it is probable that an old rheumatic process was overlooked. It will be recalled that these 19 cases were examples of primary bacterial endocarditis. In the entire necropsy series of 5215 cases there were seven cases of acute bacterial endocarditis secondary to a pneumonic process occurring, apparently, on undamaged valves.

TABLE II

DISTRIBUTION AND DEGREE OF UNDERLYING RHEUMATIC VALVULAR DAMAGE IN 35 CASES OF SUBACUTE BACTERIAL ENDOCARDITIS

Degree of Rheumatic Valvular Deformity	Number of Cases			
	Mitral Valve	Aortic Valve	Pulmonary Valve	Tricuspid Valve
I.	9	5	0	2
II.	11	9	0	0
III.	8	10	1	0
IV.	4	0	0	0

The bacterial vegetations in the group of subacute endocarditis cases were distributed as follows: mitral valve alone, 14 cases; aortic valve alone, nine; mitral and aortic valves, 19; all four valves, two; and other combinations, three cases. Thus the mitral valve was involved alone or in combination with others in 36 cases and the aortic alone or in combination, in 32 cases. These findings correspond with those of Thayer[6], Clawson[7], Blumer[8], and Fulton and Levine[4].

TABLE III

DISTRIBUTION AND DEGREE OF UNDERLYING RHEUMATIC VALVULAR DAMAGE IN 17 CASES OF ACUTE ENDOCARDITIS

Degree of Rheumatic Valvular Deformity	Number of Cases			
	Mitral Valve	Aortic Valve	Pulmonary Valve	Tricuspid Valve
I.	3	1	1	0
II.	9	5	1	1
III.	2	1	0	0
IV.	1	1	0	0

In the acute cases, the distribution of the bacterial vegetations was as follows: mitral valve alone, eight; aortic alone, six; mitral and aortic in combination, four; mitral, aortic, and tricuspid in combination, one. In three cases in which there was rheumatic endocarditis of the mitral valve, the vegetative process was present on the aortic and tricuspid valves without

evidence of an underlying rheumatic basis[1]. Thus the vegetations occurred on the mitral valve in 13 cases, on the aortic valve in 11, and on the tricuspid in one case.

ULCERATION AND HEALING OF VALVES IN BACTERIAL ENDOCARDITIS

In every case of acute bacterial endocarditis, a well-defined ulcerative destructive process was present. This finding was absent in 37 of the 47 cases with subacute bacterial endocarditis. In four cases the ulceration was slight, in two moderate and in four marked, resulting in perforation of the valves in two and in rupture of a cusp in one. The blood culture in two cases with ulceration showed streptococcus viridans, indicating that this organism alone may cause ulceration. Streptococcus hemolyticus was cultured from antemortem blood in one case. This was the probable organism in another patient with an associated terminal erysipelas. A streptococcus was obtained from the heart's blood after death in a fifth case, and a pneumococcus was cultured from the pericardium and heart's blood in a sixth.

Evidence has been accumulated that the vegetations of subacute bacterial endocarditis may heal to some extent. Cases have been reported by Weiss and Rhoads[8], in which such processes have apparently undergone complete healing. In going over the 5215 necropsy records attention was directed to any description that might suggest a healed subacute or acute lesion of the valves, and eight cases with suggestive evidence of complete healing were noted. The following abnormalities were considered to suggest a healed or healing process: (1) the presence of large irregular calcified nodules on, and removed from, the *free margin* of a valve without pronounced degenerative changes in the vessels; (2) calcified nodules with healed ulcerative areas; (3) small calcified nodules extending on to the chordae tendineae and to the endocardium; (4) the presence of calcification in a bacterial vegetation.

SUMMARY AND CONCLUSIONS

1. The incidence of subacute and acute bacterial endocarditis among 5215 consecutive necropsy examinations at the Boston City Hospital was 0.9 per cent and 0.4 per cent respectively.

A study of the interrelation between rheumatic and bacterial endocarditis revealed that approximately one case of subacute bacterial endocarditis occurred to every 10 cases of rheumatic heart disease; and approximately one case of acute bacterial endocarditis to every 25 cases of rheumatic heart disease. One case of subacute bacterial endocarditis occurred to approximately every four cases of fatal rheumatic heart disease; and one case of acute bacterial endocarditis to approximately every nine cases of fatal rheumatic heart disease.

2. Whereas rheumatic heart disease occurred more frequently among females, subacute and acute endocarditis superimposed on rheumatic heart disease developed considerably more often in males (ratio 3:1).

3. The distribution of the *degree* of rheumatic valvular damage in the 47 cases of subacute bacterial endocarditis corresponded with that for 474 cases of rheumatic heart disease, indicating that the development of subacute bacterial endocarditis is independent of the *degree* of underlying rheumatic endocarditis.

4. The apparent infrequency of advanced mitral stenosis with subacute bacterial endocarditis is explained by the fact that slight or severe "non active" rheumatic valvular lesions predispose equally to the development of subacute bacterial endocarditis; and slight lesions are much more common than advanced ones. The same factor is held responsible for the relatively rare occurrence of severe congestive heart failure and auricular fibrillation in the course of subacute bacterial endocarditis.

5. Cases with morphological evidence of varying grades of healing of the valves were encountered in subacute bacterial endocarditis.

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APPENDICITIS

A Clinical and Pathological Study

BY HENRY F. HOWE, M.D.*

GOOD-HUMORED criticism of the surgeon for the frequency of his appendectomies is not wanting among his colleagues in pathology and medicine. They sometimes ask: "What would be the findings in a hundred consecutive cases of right lower abdominal pain operated on under a diagnosis of appendicitis?" The present study is of such a consecutive series from the Harvard Surgical Teaching Service of the Boston City Hospital during the months from November, 1931 to May, 1932. During this same period forty cases complaining of right lower quadrant pain were discharged without operation with diagnoses of constipation, pyelitis, gastro-enteritis, etc. The cases in our study were twelve years of age or over. No "incidental" appendectomies in the course of operations for other diseases are included. The series is small, but is presented because the data are more complete than in many larger series. All the cases were studied and the information obtained by the writer in person. Effort has been exerted to observe uniform standards in evaluating signs, symptoms and findings. The facts have been summarized with a view to establishing: (1) a working classification of the types of appendicitis with a detailed study of "recurrent" or "chronic" appendicitis; (2) a suggestive group of diagnostic errors; (3) a correlation between operative and pathologic findings; and (4) a correlation between clinical signs and these findings.

DATA

The data are such as can easily be obtained routinely in an ordinarily thorough study of any case of right lower quadrant pain. With few exceptions the following facts were recorded:

1. Age and sex of patient.
2. History of previous similar attacks.
3. Interval from onset of present attack to admission.
4. In female patients, interval since onset of last menses.
5. Presence or absence of following symptoms in present attack: fainting; nausea; vomiting; constipation; diarrhea; generalized abdominal pain at onset; urinary symptoms; upper respiratory symptoms.
6. Presence or absence of following physical signs on admission: tenderness; spasm; rebound sign; psoas sign; tenderness by rectal or pelvic examination; palpation of a mass.
7. Temperature; pulse; total leucocyte count; white blood cells in urine.
8. Presence or absence of the following findings at operation: free peritoneal fluid; old fibrous adhesions about the appendix; pus or fibrin on the surface of the appendix; kinking or obliteration of the appendix; fecoliths in the appendix; position of the appendix as to its being retrocecal or in the pelvis; other pathology noted on exploration.
9. Type of operation.
10. Pathological report on tissue removed at operation.
11. Final Diagnosis.
12. Complications and mortality (not included in this paper).
13. Further study of microscopic sections of appendices.

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CLASSIFICATION OF CASES

Acute Appendicitis			**44 cases**
Unruptured		27	
Of these there were conditions as follows:			
Retrocecal	11		
Pelvic	5		
With fecoliths	3		
Healing	2		
Carcinoid	1		
With salpingitis	1		
Unclassified	4		
Ruptured		17	
Appendix Abscess	5		
Local Peritonitis	5		
General Peritonitis	3		
Perforated without Peritonitis	3		
Tuberculosis	1		
Recurrent Appendicitis			**26 cases**
Among these cases conditions existed as follows:			
Old Adhesions	25		
Retrocecal	13		
Kinked	6		
Fecoliths	3		
Obliterated	5		
Pin-worms	2		
Jackson's membrane	1		
Abscess of omentum	1		
Ovarian Pathology			**12 cases**
Ruptured Hemorrhagic Cysts		8	
Follicular Cysts		4	
Salpingitis			**1 case**
Ileocecal Tuberculosis			**1 case**
Upper Respiratory Infection			**3 cases**
Acute Rheumatic Fever			**1 case**
Abdominal symptoms unexplained			**12 cases**
			100 cases

The classification given above is based on a combination of the microscopic reports from the Pathological Laboratory of the Boston City Hospital with observations of the gross appearances at operation by the writer who is a former resident of that Laboratory. All the 27 cases termed "Acute Appendicitis, Unruptured" were so re-

ported by the Laboratory except two which were reported Healing Appendicitis. Of the 17 cases of "Acute Appendicitis, Ruptured," the 14 in which appendectomy was done were so reported by the Laboratory, one being tuberculous. The remaining 56 cases were uniformly reported "Healed Appendicitis" in so far as the appendix was concerned except for mention of obliteration, fecoliths, and pin-worms. Of these 56 cases, gross appearances at operation of old fibrous adhesions binding the appendix down, together with fecoliths, obliteration, kinking or pin-worms, seemed to justify in the 26 cases called "Recurrent Appendicitis" a conclusion that there was enough mechanical interference with the appendix to account for the severity of the symptoms. This group of 26 cases are of the class that constitute the moot point in discussion of the disease, particularly between the surgeon and the pathologist, but the writer's microscopic training in pathology could not altogether blind him to definite gross evidences of pathology in all these cases, although microscopically no evidences of inflammation appeared in any of these appendices.

Of the remaining 30 cases the most remarkable feature is the frequency of ruptured hemorrhagic cysts of the ovary. It is the writer's belief that this percentage of such cysts is proportionately much higher than would appear in a larger series, but their frequency in this series at least suggests that they are commoner than is ordinarily supposed. Whether lack of thorough exploration of the pelvis in routine appendectomies, or some other factor, has been responsible for failure to recognize this condition in the past is an interesting problem. Another subject for study is the observation that in seven of these eight cases rupture occurred in the right ovary. The gross appearances at operation suggested ruptured corpora hemorrhagica in seven instances, and a ruptured hemorrhagic follicular cyst in the eighth.

There were four cases in which simple follicular cysts of the ovary 1.5 cm. or more in diameter were the only finding. There had been menstrual irregularities in two of these cases, and it seemed fair to consider the symptoms referable to the ovaries in the four cases because of the size of the cysts. In two of the cases called "Unexplained" there were small grape-like clusters of thin-walled parovarian cysts. The writer does not believe that these could account for appendicitis-like symptoms.

The most significant comment to be made about the above assortment of cases is that a full 30% were not appendicitis at all, and that 18% were instances of other diseases simulating appendicitis. No more striking support could be given to the idea that it is folly to remove a negative appearing appendix without enlarging the incision sufficiently to make a thorough exploration of the abdominal cavity. To be able to show a patient a negligible abdominal scar postoperatively is never so important as the ability to tell him, or her, that in your best judgment the symptoms will not recur. An esthetic incision is a poor alibi for failure to cure disease.

DIVERGENT VIEWS OF RECURRENT APPENDICITIS

The difficulty of establishing a pathological classification of appendicitis which will correlate with the surgeon's observations clinically or at operation, arises from the difference in the situations in which the pathologist and the surgeon examine the appendix. The surgeon having struggled to free up an appendix from dense old adhesions that bind it down, kinked like a fish-hook, to the retrocecal fossa, is considerably perturbed to receive the pathologic report of "Healed Appendicitis" which is in no way distinguishable from a report on another appendix which was entirely negative in appearance. Why should there be this discrepancy? The explanation is that most pathologists rely principally on the microscopic findings for their diagnoses. The appendix arrives at the laboratory flattened out, with the checkerboard of a gauze texture well imprinted into its once glistening surface. It then becomes a difficult matter to identify adherent tags of scar in the gross examination, and if there are no evidences of a fecolith or an obliteration of the lumen, the mechanical effects of combined adhesions and kinks do not become evident to the pathologist. The microscopic section reveals no ulceration of the mucosa, no leucocytes in the muscularis, and usually no definite accumulation of lymphocytes. The adhesions are for the most part too thin to constitute an abnormal microscopic finding, even if still present and not all stripped away at operation. Consequently many pathologists adopt the blanket report of "Healed Appendicitis" and leave the issue as to whether there was pathology present to the operative record.

It becomes evident that an honest study of any series of "chronic" or "recurrent" appendicitis involves the pathologic judgment of the surgeon in making his fair or unfair estimate of the degree of abnormality in an appendix as he finds it at operation. Oftentimes he has had no pathologic training. Hence the pathologist lifts his eyes occasionally at the whole notion of chronic appendicitis. The perturbed surgeon and the skeptical pathologist combine unfortunately to throw discredit on the very existence of the disease entity. Meanwhile the patient suffers from what he considers is a very real ailment. We cannot emphasize too strongly that when the symptoms and the operative and the pathologic findings are taken together, the disease entity becomes apparent and real. These patients do have symptoms, symptoms often in-

distinguishable from full-fledged acute appendicitis. In the great majority of cases the findings at operation satisfactorily explain these symptoms, and usually justify the operative procedure entirely aside from the argument that operation prevents a later acute process in the appendix. We are convinced also that there is a very characteristic microscopic finding in many of these cases, which is sufficiently reliable to distinguish most of them from negative appendices.

THE MICRO-PATHOLOGY OF RECURRENT APPENDICITIS

Something of the dilemma that the bound down appendix presents to the pathologist has been already described. The pathologist thinks of the cardinal signs of inflammation as necrosis and leucocytic infiltration. Neither is present in these appendices. Frequently there are accumulations of lymphocytes in the lymphatic capillaries of the muscularis and serosa, but this finding is quite inconstant and often occurs in what to the surgeon appear to be entirely negative appendices. This was proved in numerous instances in this series.

We have referred above to the gross evidences of appendiceal obstruction in many of these recurrent cases. Over and over again surgical writers suggest that pain arises from mechanical obstruction and distention of the appendix by fecoliths, kinking or obliteration of the lumen. Usually the argument ends at the surgical level, however. It should not do so. If there is distention of the appendix, the microscopic picture of distention should appear in the stained section of the appendix. This is a simple problem to determine in such a series as the present one, where the operative findings are known, and the degree of probable gross obstruction can be evaluated. If there is sufficient obstruction of an appendix to produce pain, and the supposition is borne out by finding a bound down appendix, the evidences of distention should still be present microscopically even though the organ has suffered some handling.

The mechanical responses of an intestinal viscus to obstruction are twofold. Peristalsis is excited, and secretion, presumably with a view to washing away the obstruction, is increased. An obstruction of the gall bladder produces colicky peristalsis and gross hypersecretion of mucus in its early stages. In the appendix as received by the pathologist there is no very good gross or microscopic method of estimating whether peristalsis was effective in emptying the organ, and gross mucus is a rare finding in the appendix. It is easy to recognize microscopically the degree of secretory activity of the mucus glands of the appendix, however, and to make a rough estimate of intra-appendiceal pressure, of which these glands serve as a sort of barometer. The secreting cells are a single row of cylindrical goblet-cells lining straight tubular glands. Normal secretory activity is indicated by round or slightly oval clear vacuoles appearing in the cytoplasm bordering the lumen of the gland in occasional cells throughout the gland. The lumen also contains a few round or oval globules of secretion. This is the usual normal picture throughout the appendix, though occasional individual glands may normally give an appearance of distention with mucus or of absence of activity.

In going over the microscopic sections of the appendices in this series, the writer was struck by the fact that in most of the early acute appendices where the mucosa was not yet destroyed, the goblet-cells in one or more of the cross-sections of the appendix were almost universally distended by large cylindrical vacuoles extending deep into the peripheral portion of the cells, sometimes even displacing the nuclei to one side. This is the usual microscopic picture in obstruction of secreting glands. Turning then to the slides of the recurrent appendices, the findings were not so consistent, but it was recognizable that cases where the appendix, in addition to being bound down, also had a kink, an obliteration or a fecolith, usually gave the same picture of hypersecretion and distention of the glandular epithelium with mucus that the acute cases had demonstrated. Usually the lumen of the gland was almost or quite closed by distention of the goblet cells with mucus, and this finding tended to be constant throughout all parts of a section rather than merely occasional in a few glands. Such a situation certainly suggests intra-appendiceal pressure sufficiently severe and long-standing to impair temporarily the free drainage of the tubular glands, and where this appearance is universal throughout a section it seems fair to assume that the corresponding segment of the appendix is distended by something more intense and more prolonged than the pressure of normal peristalsis.

This picture of universal mucous distention was found typically in 57% of the recurrent appendicitis cases. The remainder varied all the way from an appearance of moderate distention to that of normal secretion. The 30 cases in which the appendix was presumably negative showed only occasional instances of glandular distention. No reliable control series of incidental appendectomies has yet been studied, and the only trustworthy conclusion that can therefore be drawn from these observations, is that it is possible through microscopic study of the secretory activity of the appendix to identify the most marked cases of "Obstructively Healed Appendicitis." Of the eighteen cases which showed this microscopic finding, eleven were cases with fecoliths, gross obliteration or kinking of the appendix. Mucous dis-

tention was present microscopically in 83% of the kinked appendices, all of the partially obliterated appendices, and in all of the cases with fecoliths. This should be a welcome bit of pathologic support for those surgeons who have speculatively concluded that recurrent appendicitis is often a disease of mechanical origin; it should also be considered a suggestion as to the character of the earliest stage of acute appendicitis.

CLINICAL STUDY

In the following tables the figures represent percentages of cases of each disease group rather than of the whole series. The conclusions given are in some instances to be discounted because of the small number of cases which they represent. All these conclusions apply only to differential diagnosis among the four conditions rather than between them and other abdominal conditions.

AGE INCIDENCE

	12-21 yrs.	22-31 yrs.	32-41 yrs.	42+ yrs.
Acute Ruptured	41	12	18	29
Acute Unruptured	59	29	4	8
Recurrent	58	23	15	4
Ruptured Hemorrhagic Cyst—100% aged 15 to 25				

Conclusions:

1. Ruptured acute appendicitis tends to occur most frequently between the ages 12-21 and in adults above 40.
2. The incidence of both acute and recurrent appendicitis is greatest in the age group 12-21.
3. Ruptured hemorrhagic cysts of the ovary tend to be confined to the ages 15-25.

PREVIOUS ATTACKS AND BOUND-DOWN APPENDICES

	Prev. Att.	No Prev. Att.	Adhesions	No Adhes.
Acute Ruptured	12	88	29	71
Acute Unruptured	33	66	22	78
Recurrent	56	44	96	4

Conclusions:

1. Both ruptured and unruptured acute appendicitis tend to occur in the first attack in a nonadherent appendix.
2. There is a tendency to direct correlation between the ability to elicit a history of previous attacks and the operative finding of old fibrous adhesions about the appendix.

NAUSEA AND VOMITING

	Nausea	No Nausea	Vomiting	No Vomiting
Acute Ruptured	88	12	71	29
Acute Unruptured	81	19	78	22
Recurrent	70	30	39	61
Rupt. Hemorrh. Cyst	62	38	50	50

Conclusions:

1. Nausea is characteristic of all forms of appendicitis and of ruptured hemorrhagic ovarian cysts, but vomiting suggests acute appendicitis.
2. Vomiting occurs, however, in from a third to a half of the recurrent appendicitis and hemorrhagic cyst cases also.

CONSTIPATION AND DIARRHEA

	Constip.	No Constip.	Diarrhea	No Diarrhea
Acute Ruptured	44	56	0	100
Acute Unruptured	52	48	28	72
Recurrent	54	46	0	100
Rupt. Hemorrh. Cyst	25	75	25	75

Conclusions:

1. Constipation is of no assistance in differential diagnosis of these four conditions.
2. Gastro-enteritis being ruled out, diarrhea sometimes suggests acute appendicitis.

URINARY SYMPTOMS AND WBC IN URINE

	G. U. Sympt.	No G. U. Sympt.	WBC	No WBC
Acute Ruptured	12	88	27	73
Acute Unruptured	4	96	19	81
Recurrent	13	87	8	92
Rupt. Hemorrh. Cyst	25	75	0	100

Conclusions:

1. Urinary symptoms are occasional in all these cases.
2. In only 7% of the retrocecal cases were there urinary symptoms. One of these was a ruptured acute and the other a recurrent appendicitis. A different 7% of the retrocecal appendices showed leucocytes in the urine, both these cases being acute appendicitis.

UPPER RESPIRATORY SYMPTOMS

	Cold, Sore Throat	No Cold or S. Thr.
Acute Ruptured	29	71
Acute Unruptured	4	96
Recurrent	21	79
Rupt. Hemorrh. Cyst	13	87

Conclusion:

1. Upper respiratory infections are frequent in both acute ruptured and recurrent appendicitis.

TIME FROM ONSET TO HOSPITAL ADMISSION

	1-12 hrs.	12-24 hrs.	24-48 hrs.	2-4 days	4-7 days	7 days +
Acute Ruptured	12	12	48	6	6	18
Acute Unrupt.	29	26	26	15	4	0
Recurrent	8	4	8	20	20	40
Rupt. Hemorrh. Cyst	37	37	0	13	0	13

Conclusions:

1. An acute appendicitis which ruptures does so most frequently between 24 and 48 hours after the onset of symptoms, but may do so as early as five hours.
2. All acute cases that entered more than one week after onset had formed appendix abscesses.
3. Acute unruptured appendicitis patients tend (in Boston) to enter a hospital within 48 hours after onset. Those with recurrent attacks tend to apply for admission at a longer interval than two days after onset, and most frequently after a week of symptoms.
4. Patients with ruptured hemorrhagic cysts of the ovary usually enter within 24 hours of onset.

GENERALIZED PAIN PRECEDING RLQ PAIN

	Generalized Pain	No Generalized Pain
Acute Ruptured	87	13
Acute Unruptured	85	15
Recurrent	40	60
Rupt. Hemorrh. Cyst	50	50

Conclusion:

Generalized abdominal pain preceding the right lower abdominal pain usually suggests acute appendicitis although it is frequent also in recurrent appendicitis and ovarian hemorrhage.

SPASM AND REBOUND SIGN

	Spasm	No Spasm	Rebound	No Rebound
Acute Ruptured	100	0	71	29
Acute Unruptured	100	0	79	21
Recurrent	35	65	16	84
Rupt. Hemorrh. Cyst	63	37	71	29

Conclusion:

Positive muscular rigidity and rebound sign are usually coincident, and when present are almost diagnostic of acute appendicitis, but are usually present also in cases of ruptured hemorrhagic ovarian cysts. It is this similarity which makes the two diseases so difficult to distinguish.

PSOAS SIGN AND TENDERNESS BY RECTUM

	Psoas	No Psoas	Rectal Tend.	No Rect. Tend.
Acute Ruptured	57	43	69	31
Acute Unruptured	40	60	85	15
Recurrent	5	95	50	50
Rupt. Hemorrh. Cyst	25	75	86	14

Conclusions:

1. A positive psoas sign is useful only as a confirmation of a rather severe degree of acute appendicitis. Its occasional occurrence in ovarian hemorrhage is again confusing.
2. Right-sided tenderness by rectal or pelvic examination is consistent with any one of these four conditions, but is most suggestive of acute appendicitis or ruptured corpus hemorrhagicum.

TOTAL LEUCOCYTE COUNT

	5-10000	11-14000	15-19000	20000+
Acute Ruptured	0	18	41	31
Acute Unruptured	4	19	58	19
Recurrent	30	43	19	8
Rupt. Hemorrh. Cyst	25	50	25	0

Conclusions:

1. A white count of over 15000 is strongly suggestive of acute appendicitis, although one acute case in the series showed only 5000.
2. A white count of less than 15000 suggests recurrent appendicitis or ovarian hemorrhage, although counts of 20000 or above do occur in the former.
3. Counts of 20000 or above usually indicate a gangrenous or ruptured appendix, in the absence of any additional active focus of infection.

PULSE

	60-80	81-99	100+
Acute Ruptured	12	12	76
Acute Unruptured	14	55	31
Recurrent	33	38	29
Rupt. Hemorrh. Cyst	25	50	25

Conclusions:

1. The pulse taken in the admitting room is of very little value unless judged in association with the personality and emotional state of the patient.
2. A pulse of over 100, subject to the above qualification, is quite suggestive of a ruptured appendix.

TEMPERATURE

	97-98.9	99-99.9	100+
Acute Ruptured	16	16	68
Acute Unruptured	0	66	34
Recurrent	20	80	0
Rupt. Hemorrh. Cyst	0	100	0

Conclusions:

1. The great majority of admission temperatures fall between 99 and 100 degrees, and no reliable conclusions can be drawn from this group.
2. Temperatures above 100 suggest acute appendicitis, probably ruptured, unless there is some additional infectious focus.

GROSS PATHOLOGY OF APPENDIX

	Kinking	Obliteration	Fecoliths	Adhesions
Acute Ruptured	0(?)	0(?)	6	20
Acute Unruptured	4	7	11	22
Recurrent	24	20	12	96
All other Cases	3	3	0	3

Conclusions:

1. Kinking, obliteration and fecoliths of the appendix occur occasionally in acute appendicitis, but much more frequently in recurrent appendicitis. They are definitely more frequent in both than in the average run of cases subjected to laparotomy.
2. Acute cases with fecoliths tend to show a higher white count than other acute cases.
3. Only about a fourth of acute cases, whether ruptured or not, occur in appendices bound down by old adhesions.
4. Of the cases which show definite gross evidence at operation of old fibrous adhesions about the appendix, 58% show also gross obstruction by fecoliths, obliteration or kinking. This 58% is almost identical with the 57% that show microscopic evidence of mucous distention.

RETROCECAL AND PELVIC POSITIONS OF APPENDIX

	Retrocecal	Pelvic	Neither Ret. Nor Pel.
Acute Ruptured	29	12	59
Acute Unruptured	41	18	41
Recurrent	50	0	50
All other cases	6	6	88

Conclusions:

1. Both retrocecal and pelvic positions of the appendix are vastly more frequent in diseased appendices than in those removed incidentally to other operations.
2. The retrocecal appendix appears to be more susceptible to pathology than that situated in the pelvis.

OTHER DATA OBSERVED

There was no observable correlation in any of these cases, ovarian included, with any particular portion of the menstrual cycle. The corpora

hemorrhagica varied at admission from one day after onset of the menses to one day previous to onset, and there was no tendency of the cases to be grouped in any particular phase of the cycle.

Sex was not found to be an important factor in acute or recurrent appendicitis, but the unexplained cases were more commonly females than males.

In only one case of ovarian hemorrhage was there a history of fainting at onset. One of the acute appendicitis cases also gave such a history.

It is interesting to observe that only two of the 44 acute cases were reported pathologically as in the healing stage, in view of the fact that 17 were ruptured. This is susceptible to two interpretations: (1) that the possibility of recovery from acute appendicitis without operation lies most commonly in resolution of a localized peritonitis or abscess rather than in healing of the appendiceal process without rupture; or (2) that those cases which would heal without rupture nowadays come to operation before a healing stage has begun. If the first alternative is accepted, we must assume that most of the 26% of recurrent appendicitis cases went through the stages of rupture and resolution of an abscess in the course of healing. The character of the histories of previous attacks, and the type of adhesions in which most of these recurrent appendices were found, belie this theory of healing, and the writer believes rather that many of our acute cases would heal without rupture if not operated on, and pass on into the secondary or recurrent stage of appendicitis.

EXCEPTIONS

There is no disease in which the average picture can lead one farther astray in diagnosis than appendicitis. No more than two or three out of the 44 acute cases cited could be said to be textbook illustrations of acute appendicitis. For this reason tables of averages such as the above are gross misrepresentations unless some of the striking exceptions are noted along with them. The table which appears below will serve to illustrate some of these:

SUMMARY

Data collected in one hundred consecutive cases operated on with a diagnosis of appendicitis by the Fifth Surgical Service of the Boston City Hospital are presented, together with a discussion of the pathological and clinical aspects of the disease which they suggest.

The following working classification of appendicitis is suggested: 1. Acute Appendicitis, Ruptured, 2. Acute Appendicitis, Unruptured, 3. Recurrent Appendicitis, the last-named to include those cases which by a combination of operative and pathologic examination can fairly be said to contain such pathology as, by obstruction or interference with the proper emptying of the appendix, should cause right lower abdominal pain. Microscopic evidence is given in support of the notion that such obstruction exists in over half of these cases, in the form of mucous distention of the tubular glands. The clinical data indicate that a history of previous attacks and nausea without vomiting in a patient who has had symptoms for more than 48 hours in the right lower abdomen without appearing acutely ill, together with a white count of 11-14,000, no spasm or rebound sign, but marked tenderness, suggests the diagnosis of recurrent appendicitis.

The data do not include follow-up notes to determine whether the operative treatment gave permanent relief of symptoms. The classification is suggested as an attempt to clarify the group of pathological conditions usually termed chronic appendicitis under a reasonable physiological etiology judged by microscopic evidence.

The data are so marshaled as to bring out

Name	Age	Sex	Previous Attacks	Time Since Onset	Generalized Pain	Nausea	Vomiting	Constipation	Diarrhea	Urinary Symptoms	Respir. Symptoms	Pulse	Spasm	Rebound	Psoas Sign	Rectal Tenderness	WB Count	Urinary WBC	Final Diagnosis
H. F.	16	F.	0	24h	+	+	+	+	0	0	sore throat	96	+	+	+	+	22	0	Recurr. Append. Retrocecal.
J. V.	38	M.	0	4d	0	0	0	0	0	0	0	80	+	+	0	+	5	0	Acute Append. Gangrenous.
M. S.	17	F.	0	2d	+	+	+	+	0	dys ur	0	80	+	+	+	+	10	0	Ac. Rh. Fever.
D. C.	18	F.	0	14d	+	+	+	+	0	0	0	80	+	+	+	+	10	0	Unexplained.
McC.	19	F.	?	3h	0	0	0	+	0	dys ur	0	112	+	+	0	+	11	0	Rupt. Hemorrh. Cyst.

certain useful diagnostic principles such as should be useful in the examining room.

A confusing group of cases of ovarian hemorrhage is presented and compared with the cases of appendicitis.

It is demonstrated that the diagnosis of appendicitis is about 70% accurate, and that in about 12% of cases laparotomy does not reveal the diagnosis.

Finally, the observations in this series tend to confirm the idea that appendicitis is usually a recurring disease. Furthermore they suggest that it is ordinarily a disease of primary acute and secondary recurrent phases. Only occasionally is the secondary phase also acute. Inasmuch as more than a third of the operated cases of the disease are in the recurrent phase, however, it is obvious that many primary acute attacks still go unrecognized, or at least unoperated. Whether operation in all the recurrent cases here cited was necessary at the time is debatable, but that in all these cases a recurring disease was checked at the source is undeniable.

ATROPINE AS A DIAGNOSTIC AID IN APPENDICITIS

BY FRANCIS M. FINDLAY, M.D.*

THE progressive increase in the incidence and mortality of appendicitis during the twentieth century has been the just cause of great concern in the minds of surgical leaders the world over. It is a reflection on our profession that the United States with over seven thousand well-equipped hospitals, adequate transportation facilities and an abundance of well-trained surgeons in nearly every community should lead the world in deaths from this acute abdominal disease and should have a mortality rate twice that of England and Wales. The Metropolitan Life Insurance Company estimates that during the past three decades the mortality from appendicitis in the United States has increased more than 50 per cent until it now annually claims as victims over twenty-five thousand persons and ranks second only to cancer as a cause of death from surgical diseases. The incidence of acute appendicitis is greatest between the ages of ten and thirty. When we realize that the majority of deaths from this disease occurs among individuals in the prime of life, the tragedy becomes even greater.

Lack of interest on the part of the profession cannot be held entirely responsible for this state of affairs. Scarcely a year passes without the subject being discussed in every local and state medical society while over 2500 articles on appendicitis appeared in the world's literature in the past decade. The importance of early operation and the dangers inherent in delay have been heralded among the profession. Both layman and physician have been taught that it is criminal to use cathartics or opiates in the presence of undiagnosed abdominal pain. Four years of organized publicity warning against the dangers of indiscriminate use of laxatives lowered the mortality rate 16% in Philadelphia.

Wilkie has pointed out that the acute disease is of two distinct types, the one an infective inflammation of the wall of the appendix, the other a sudden obstruction of the lumen of the organ with the blocking of the toxic appendiceal contents—really a form of intestinal obstruction. The clinical picture in the first type is that of an inflammatory lesion within the abdomen, with the well-known symptoms of increased pulse and temperature, vomiting, pain and local tenderness which render diagnosis in the vast majority of cases relatively easy.

The picture in the second type is totally different, for here we have the sudden onset of spasms of pain, usually with vomiting, without any increase in temperature or pulse rate. For the first few hours the patient does not appear to be acutely sick and except for the recurring colic does not complain. Only careful examination will disclose the sharply localized tenderness that is diagnostic. Necrosis and perforation proceed at a rapid rate in this obstructed type, and often there is a diffuse peritonitis in less than twenty-four hours from the onset of the first symptom. Since 90 per cent of all deaths from appendicitis occur in this rapidly fulminating type of case, we must make every effort to diagnose and operate on this group of cases before perforation takes place. We must emphasize the acute emergency presented by this second group and render its early diagnosis more certain.

What diagnostic points deserve more careful consideration? The late John B. Murphy first stressed the importance of the proper sequence of symptoms in making a diagnosis of acute appendicitis. Abdominal pain, followed by nausea and vomiting with a subsequent area of local tenderness over McBurney's point is the classical order of events. Rigidity, fever and leukocytosis usually follow in rapid order in the first type of appendicitis. The findings vary and the clinical picture is often very different in the second type. Bower, in his study of both types of the disease in Philadelphia, found that pain was the only constant symptom. The history of sudden onset of cramp-like abdominal pain should always be sought. The significance of

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recurring colic, however mild, cannot be overlooked if we are to improve our early diagnosis in this second type, the obstructive form of acute appendicitis.

Of all the symptoms and signs found in both types of acute appendicitis, pain and tenderness are the most difficult to measure and evaluate. One patient will complain of severe pain; another passes lightly over it and tends to disregard it. Both may have an acute gangrenous appendix or, on the other hand, only an acute gastro-enteritis. How can we differentiate these conditions in the early stages? Are there any further diagnostic aids?

We have found the use of atropine to be of great value in differentiating the pain and tenderness due to acute appendicitis and that due to other conditions. As we find no mention of the routine use of atropine as a diagnostic aid in appendicitis in the literature, we take this opportunity to record our experience, and trust that it may be of value to others.

Atropine sulphate has been very widely used in this country as a medication given preliminary to the administration of an inhalation anesthetic. Its power to prevent and arrest excessive mucous secretion in the upper respiratory passages has been well known for many years. The ophthalmologists have taken advantage of its power to dilate the pupil and the pediatricians and a few internists have used it in the treatment of spasmophilia, but the profession as a whole has not fully realized its value as an antispasmodic and as a diagnostic aid in intestinal colic. To our knowledge, Dr. Marshall Bailey was the first to use this drug in diagnosis although others may rightly have prior claim. In any suspected case of appendicitis, the patient, if an adult, is given 1/100 grain of atropine sulphate. The drug is given by mouth unless the patient is nauseated when it is given by the subcutaneous route. One half hour later he is given an enema of two quarts of warm soap suds. After the enema is expelled the abdomen is carefully palpated and if an area of distinct localized tenderness persists in the region of the appendix a positive diagnosis should be made. If the enema has been only partially expelled and the entire cecum feels full or emits a gurgling sound on palpation, the atropine and enema may be repeated to ensure a thorough emptying of the large bowel. If either pain or local tenderness persists after this procedure we feel justified in making a diagnosis of appendicitis.

We have used this method for four years in a series of over 300 cases complaining of acute abdominal pain. Of the 86 cases operated upon on the basis of the test, all but one have shown definite evidence of acute appendicitis. Of the cases with negative findings, which were not operated upon, none developed acute appendicitis. Patients with food poisoning, infectious colitis, and other forms of gastro-enteritis have been given prompt relief from their discomfort by the administration of atropine which has often been repeated two or three times.

In the first type of appendicitis the onset of pain is usually very mild and the pain gradually increases in severity so that by the time the doctor is called the patient is conscious of definite pain and will admit it. In early diagnosis of this type we have found the use of atropine helpful in differentiating the colic due to enteritis, colitis or food poisoning and that due to a real inflammatory process. Pain and tenderness from appendicitis persist while discomfort due to other causes disappears after the intestinal tract is emptied.

It is in diagnosis of the obstructive type of appendicitis, however, that we find atropine to be of the greatest value. Sudden onset of pain in this type of case is usually the initial symptom. This is caused by the sudden complete obstruction of the lumen of the appendix with rapid shutting off of the blood supply. This pain, however, is often very transitory and when the patient is seen a few hours later he will deny having any pain and only the most careful abdominal and rectal examinations will elicit the sharply localized tenderness that is diagnostic. Persisting tenderness over the appendix in an adult male after the use of atropine and an enema should never be disregarded. If the tenderness has been preceded by sudden onset of pain, appendicitis is present in at least 90 per cent of the cases.

There are three variable diagnostic signs and symptoms which should be emphasized in the diagnosis of appendicitis. They are nausea, fever and tenderness. Distinct nausea and vomiting are not always present in an acute attack of appendicitis and for this reason an attack is sometimes overlooked when the patient is first seen. If the patient has not taken food or drink for several hours, his stomach will probably be empty and he will not vomit. It may be extremely difficult to obtain even an admission of anorexia. This is especially true of vigorous young adults who have a rather fat mesentery, and omentum which covers the appendix and protects it from direct contact with the sensitive parietal peritoneum. The retrocecal appendix often is so covered as to give very little peritoneal irritation. Such individuals will often flatly deny having any nausea and only careful direct questioning reveals that they have eaten nothing for the previous twenty-four hours. How often the medical man and layman alike maintain, "He has not vomited; he cannot have appendicitis." People with a history of sudden onset of cramp-like abdominal pain should be carefully investigated for other evidences of acute appendicitis even though they have not vomited. This is true in both the in-

flammatory and the obstructive types of the disease.

It is not always appreciated that fever is uncommon in the early stages of acute appendicitis. Royster states that with the onset of an acute attack the temperature is not elevated, but before thirty-six hours have elapsed a rise will be noted. In the last 50 cases of acute appendicitis operated upon in private practice, we have been impressed by the absence of temperature in eight cases. In two cases with gangrene the temperature was subnormal. The usual temperature in the early stages of inflammatory appendicitis is less than 100° Fahrenheit. In obstructive cases the temperature as a rule is normal or subnormal. The temperatures taken by rectum are always more reliable, especially in children. The absence of fever should never prevent one from making a diagnosis of acute appendicitis if other symptoms and signs are present. If we are to reduce mortality we must often operate in the absence of fever.

By far the most valuable diagnostic sign is localized tenderness over the inflamed appendix. At times this is best found by abdominal palpation, although, with a retrocecal or pelvic appendix, rectal examination is of greater aid. Both abdominal and rectal examinations should always be done. Frequently the examiner is unable to elicit sharply localized tenderness because of a generalized sense of resistance throughout the abdomen. This is especially true when the attack has begun with rather marked retching and vomiting and the patient's entire abdomen is sore and held firmly. It is also very difficult to teach athletic young men with well-developed abdominal muscles to relax. Often the ingestion of alcohol in various forms masks the disease and complicates the picture, making abdominal examination of the utmost importance. The use of atropine followed by an enema has been of the greatest help to us in these cases, for the voluntary spasm disappears, the patient relaxes and true tenderness, if present, can be well localized. In the obstructive type of appendicitis in the early stages, tenderness is often the only positive physical finding and failure to detect it spells disaster.

Fear of operation often deters the patient from seeking medical advice early, while just fear of a mistaken diagnosis with an unnecessary operation often hinders the medical man and younger surgeon in making an early diagnosis. If we are to operate early and reduce the mortality so largely caused by this rapidly fulminating type of case, we must abolish this fear and use every aid to make early diagnosis more certain.

SUMMARY

The gradual increase in mortality from appendicitis is largely due to delay in operation. Much of this delay may be attributed to failure to recognize early the obstructive type of appendicitis. Symptoms are variable in this type and pain is the only constant symptom. The use of atropine sulphate is a distinct aid in determining the presence of local tenderness early in the course of the disease. Definite nausea and vomiting frequently do not occur in the disease, and absence of fever is common.

BETTER GAS ANESTHESIA

The Carbon Dioxide Absorption Method

BY PHILIP D. WOODBRIDGE, M.D.*

WASTEFULNESS OF OLD METHOD

THE rôle of oxygen. With the usual method of administering anesthetic gases such as nitrous oxide or ethylene, with or without ether, a continuous or intermittent flow of the gas mixed with oxygen is supplied to the patient throughout the course of anesthesia. As Waters[17, 18] has pointed out, the oxygen serves two purposes: the first, to support life by meeting the needs of oxidation of the body, and the second, to dilute the anesthetic agent. The first function is to prevent asphyxia, the second, to prevent too powerful an action of the anesthetic gas. Because oxygen is used to perform both functions, they are frequently confused; nevertheless they are really quite distinct. The diluting could probably be done with any inert gas as well as with oxygen. Nitrous oxide is so weak an anesthetic agent that more than sufficient oxygen to prevent asphyxia is almost never needed. In fact in giving nitrous oxide unsupported by ether or sedative drugs, it is difficult or impossible to give enough oxygen to supply fully the body's metabolic requirements without diluting the gas so much that anesthesia is interfered with[6, 7, 10, 13]. On the other hand, when nitrous oxide or ethylene is combined with ether or avertin or with sedative drugs in large doses, or again when a more powerful gas such as acetylene is used, then an amount of oxygen merely sufficient to keep the patient pink and to meet his metabolic needs will frequently permit too deep anesthesia to develop. Under these conditions additional oxygen must be given to perform the second function—that of further diluting the anesthetic agent.

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Fractional rebreathing. In the usual administration of gases they pass from the mixing chamber of the machine to the rebreathing bag. With some machines this is attached directly to the mask, with others it is fastened to the body of the gas machine and connected to the mask by a tube of such large caliber that the patient may easily rebreathe through it. The bag, the mask, and the large connecting tube, if any, I shall refer to as the reservoir. A constant flow of fresh gas is supplied to the reservoir, the patient inhales from and exhales into the reservoir, and the excess gas passes out through an escape valve on the mask. It is often thought that the size of the aperture in the escape valve controls the amount of rebreathing. In the long run, however, this is not true. The amount of rebreathing depends rather on the volume of flow of gas from the machine to the reservoir. If this flow is as great as the patient's respiratory volume, there will be practically no rebreathing (Fig. 5A). If the minute volume flow from the machine is half the minute respiratory volume, half of each inhalation will be of rebreathed gas; if the flow is one-quarter the minute respiratory volume, there will be three-quarters rebreathing (Fig. 5B). Such conditions are referred to as fractional or partial rebreathing. The size of the aperture in the escape valve merely controls the pressure in the reservoir.

Need of constant flow. The question arises: How rapidly shall gas be made to flow into the reservoir? If its rate were to equal the minute respiratory volume, breathing would be very quiet because no carbon dioxide would be inhaled and gas anesthesia would be very expensive because of the large amount of gas used. With the degrees of fractional rebreathing ordinarily used, the cost varies from $1.50 to $5.00 an hour. Occasionally anesthetists employ complete rebreathing for a few minutes at a time for the sake of economy (Fig. 5C). This is done by closing the escape valve and shutting off completely the flow of gases from the machine for a limited period. During this time the patient gradually exhausts the oxygen in the rebreathed mixture and replaces it with carbon dioxide (Fig. 5D). Anesthesia is not lightened because the anesthetic gas (nitrous oxide or ethylene) in the reservoir stays in equilibrium with that in the blood; but anoxemia and hyperpnea progressively increase. The anesthetist is forced to resume the administration of gas by two factors: lack of oxygen and accumulation of carbon dioxide.

Herein lies the key to the answer to the question of how rapidly gas shall be supplied from the machine. It must flow fast enough to prevent depletion of oxygen and undue accumulation of carbon dioxide in the reservoir. The depletion of oxygen can be prevented merely by supplying oxygen. Why need the anesthetic gas also be supplied? For no other purpose than to meet the second requirement, namely, to wash out the carbon dioxide. *The sole function of the additional anesthetic gas supplied throughout the maintenance of anesthesia is to flush the accumulating carbon dioxide out of the reservoir.* A constant flow of nitrous oxide or ethylene is not needed. The gas already in the patient's blood, held there by being in equilibrium with the gas in the reservoir, is sufficient to maintain anesthesia.

Cost of constant flow. The actual cost of flushing out this carbon dioxide with 90 per cent nitrous oxide and 10 per cent oxygen when the patient's respiratory volume is 12 L. (3.2 gals.) per minute would be $1.80 per hour if half rebreathing were used, $2.70 if one-quarter rebreathing were used, and $3.60 if no rebreathing were used. With a respiratory volume of 8 L. (2.1 gals.) per minute, the cost would range from $1.20 to $2.40 per hour, and with a volume of 20 L. (5.3 gals.) from $3.00 to $6.00.*

Cost of carbon dioxide absorption. The carbon dioxide can, of course, be much more cheaply removed by chemical means. Fifty cents' worth of soda-lime (sodium and calcium hydrate) introduced into the reservoir system will absorb the carbon dioxide produced during 6 to 10 hours of anesthesia. To the $.09 or less per hour which the soda-lime costs should be added $.08 to $.15 for the oxygen required by the body (200 to 400 cc. per minute) making a maximal total of $0.24 per hour for maintenance of anesthesia when there is no leak in the apparatus or beneath the mask. With a leak as great as 1 L. per minute (16 gals. per hour) the cost would be increased to but $0.57 per hour. The possible saving in expense is seen to be very large, and is but one of several advantages of the method to be described.

APPARATUS AND METHOD OF USE

History. The principle of absorbing carbon dioxide from exhaled anesthetic gases was used by Jackson[11] on laboratory animals in 1915. The first report of its application to clinical anesthesia was that of Waters[15] in 1924. In the following year Gauss[6] in Germany described his circuit apparatus for acetylene. His report, as also that of Wienecke, Wirth, and Kirchner[19], indicates that the Germans have made only partial use of the method. They have reduced gas consumption 75 per cent, but insist on a flow of about 2 L. per minute in order to make sure of maintaining the desired proportion of gases in the reservoir as well as to compensate for leaks. Today the most familiar application of the prin-

*These figures are based on a price of $4.60 for the size D (250 gal., = 947 L.) nitrous oxide tanks and $1.92 for the same sized oxygen tanks (80 gals., = 303 L.). With the very large tanks (G) the cost would be slightly over half as great.

ciple is in portable basal metabolism machines.

To-and-fro apparatus. Waters' apparatus[15, 16, 17, 18] is shown in Figure 1. The canister contains 500 Gm. of soda-lime and measures 8 by 12 cm. It is introduced between the mask and

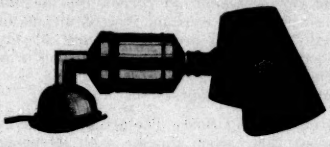


FIG. 1. *To-and-fro apparatus*, carbon dioxide absorption. Fresh gases enter at the nipple above the mask. Slip-joints at each end of the canister permit it to be removed when desired, and the bag to be attached directly to the chimney of the mask. (Photo from The Foregger Co.)

the breathing bag, so that with each respiratory cycle the gases pass through the soda-lime twice: once during exhalation on their way to the bag and again during inhalation on their return to the patient. This is therefore called the to-and-fro method. A rubber tube carries the fresh gases from the machine into the reservoir system through the connection seen between the mask and the canister. The canister is made with a slip joint at each end so that it may be removed from the system when one desires to stimulate respiration by allowing carbon dioxide to accumulate.

With this type of apparatus a full-sized bed pillow is placed beneath the patient's head instead of the usual small operating-table pillow. For induction the canister is removed from the apparatus and the mask attached directly to the breathing bag (Fig. 5E). The mask is placed upon the patient's face with the gases flowing at the rate and in the proportion to which the anesthetist is accustomed. Two or three times during induction the mask should be raised slightly from the face and the contents of the bag expressed, in order that the nitrogen previously in the patient's respiratory tract may

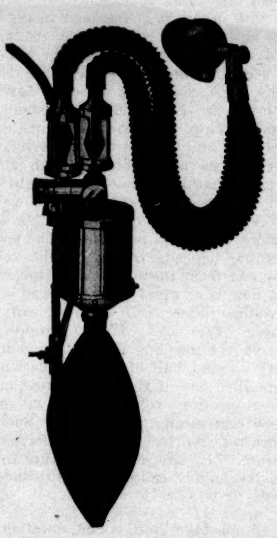


FIG. 4. *Circuit apparatus*, carbon dioxide absorption. The one-way rubber flutter valves are seen inside the glass cages above the soda-lime canister. Fresh gases enter above the inhaling valve through the small rubber tube at the left. Below these is a valve for directing exhalations either through or around the soda-lime. The bracket is of adjustable height. (Photo from The Foregger Co.)

be flushed out. The mask is then fitted very snugly to the face and fastened with a retaining strap passed behind the head. When the res-

FIGS. 2 and 3. *To-and-fro apparatus*, carbon dioxide absorption. A full-sized bed-pillow is used, and patient's head is turned to one side. The weight of the soda-lime and mask and the retaining strap around the head hold the mask securely in place. (Photos from Dr. Ralph M. Waters.)

pirations are regular and fairly deep the bag is disconnected from the mask during an expiration and the soda-lime canister is inserted between them. The patient's head is turned slightly to one side, so that the end of the canister rests on the pillow and its weight helps to hold the mask in close contact with the face (Figs. 2 and 3). The anesthetic gas (either nitrous oxide or ethylene) may then be shut off entirely and the flow of oxygen reduced to the probable metabolic need of the patient. This is usually between 200 and 400 cc. per minute. From this time on, the anesthetic gas in the reservoir remains in equilibrium with that in the patient's blood, the excreted carbon dioxide is removed by the soda-lime, and the oxygen used by the patient is replaced by the slow steady flow from the machine (Fig. 5G). If all connections are tight and oxygen is supplied at the correct rate, no further adjustments will be needed throughout the rest of the operation and no more of the anesthetic gas will be required.

Circuit apparatus. A different type of apparatus for obtaining the same result has been described by Foregger[2] and by Sword[14] (Fig. 4). The soda-lime canister is removed from the vicinity of the patient's head to the gas machine, and is connected to the mask by two rubber tubes of large caliber. One-way rubber flutter valves of very sensitive action are interposed between the canister and the large rubber tubes. These are seen in the illustration inside the cylindrical glass housings. They control the direction of gas flow, so that the expired gases pass from the face mask through the expiratory tube and valve and through the soda-lime into the breathing bag; during inspiration the gases pass from the bag around (not through) the soda-lime, then through the inspiratory valve and tube to the face mask. This is therefore called the circuit, or closed circle, type of apparatus. By means of a shunt valve the anesthetist can cut the soda-lime out of the circuit whenever he desires to stimulate respiration by allowing carbon dioxide to be built up in the reservoir system. Fresh gases from the machine enter the reservoir through the small rubber tube attached just above the inspiratory flutter valve.

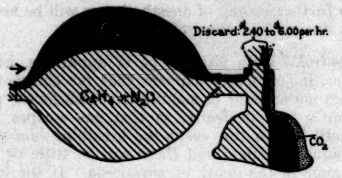
The operation of the circuit apparatus is very similar to that of the to-and-fro apparatus. For induction the soda-lime is excluded from the circuit, the anesthetic gas and oxygen are started flowing in the proportion and at the rate to which the anesthetist is accustomed, the escape valve is opened slightly, and the mask applied to the patient's face (Fig. 5F). Induction with this apparatus is therefore precisely similar to that with the ordinary type of apparatus, and ether, if desired, is similarly added. After five to ten minutes when a satisfactory and evenly maintained plane of anesthesia is reached, the anesthetist shifts to the carbon dioxide absorption technique. To do this he turns the shunt valve, which throws the soda-lime into the circuit, closes the escape valve, shuts off the flow of anesthetic gas, and reduces the flow of oxygen to between 200 and 400 cc. per minute (Fig. 5H). As with the to-and-fro apparatus, if all connections are tight and if oxygen is flowing at the correct rate, no further adjustment and no further supply of anesthetic gas will be needed during the course of anesthesia.

Control. With either type of apparatus, however, it frequently happens that the anesthetist does not succeed in making the mask fit the patient's face perfectly, so that gas escapes beneath it. In this event the breathing bag will gradually deflate and there will be little or no change in the depth of anesthesia. If the leak cannot be stopped, a slow flow of the anesthetic gas should be used, sufficient to keep the bag inflated. To this gas, should be added oxygen for dilution over and above the 200 to 400 cc. per minute needed for metabolic requirements. Usually 1 L. or less of anesthetic gas per minute (16 gals. per hour) with 100 cc., more or less, (1.6 gals. per hour) of additional diluting oxygen will suffice to compensate for the leak. Again, it may happen that, with the anesthetic gas entirely shut off, the flow of "metabolic" oxygen will prove insufficient for the patient's needs, so that he gradually exhausts the diluting oxygen. In this event the bag will slowly deflate, anesthesia will deepen, and cyanosis will develop. The obvious remedy is to restore the diluent oxygen immediately by a brief, rapid flow and to increase slightly the steady flow of "metabolic" oxygen. On the other hand, it may happen that the estimated flow of "metabolic" oxygen is too great. In this event it takes on the rôle of diluent oxygen, anesthesia lightens, and the bag becomes distended. To overcome this situation a brief, rapid flow of the anesthetic gas is used, the excess volume in the bag is reduced by opening the escape valve momentarily, and the flow of "metabolic" oxygen is reduced. If at any time it is desired to deepen anesthesia, additional anesthetic gas may be given (Fig. 5: I, J), or, if it is desired to lighten anesthesia, additional diluting oxygen may be given (Fig. 5: K, L). The resulting changes can be made to occur very rapidly, for both types of apparatus are so designed that the fresh gas passes directly to the mask without first mixing with the total volume in the reservoir.

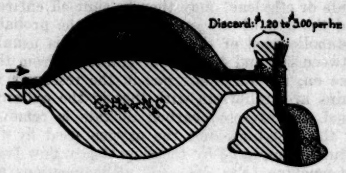
Relative advantages of the two types of apparatus. Waters' to-and-fro apparatus presents several advantages over the circuit type. It is easily portable and is easily disconnected from one machine and attached to another. For these reasons it is certainly to be preferred by the anesthetist whose work is distributed among several

hospitals*. Its initial cost is less than that of the circuit apparatus. It has fewer joints to leak, and offers less resistance to respiratory ebb and flow of gas. If the soda-lime becomes exhausted during an operation, another canister

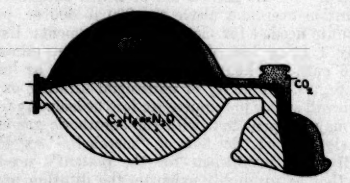
FIG. 5. DISTRIBUTION OF GASES (DIAGRAMMATIC) IN THE RESERVOIR SYSTEMS. The changes in the relative proportions of gases are shown, not their absolute quantities.



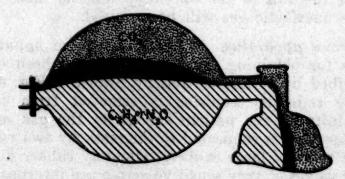
A. *No rebreathing—rapid flow of gas.* Flow of gas equals or exceeds the respiratory volume. All exhaled gas passes out the escape valve. Notice cost of discarded gas.



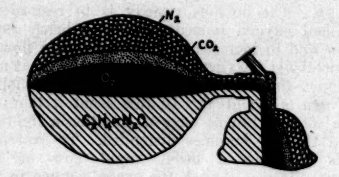
B. *Partial rebreathing.* Flow of gas is less than the respiratory volume. Part of the exhaled carbon-dioxide-laden gas passes out through the escape valve, and part returns to the bag and is re-inhaled.



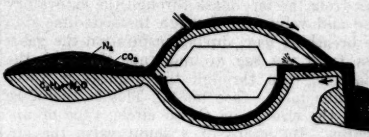
C. *Total rebreathing—onset.* Flow of gas shut off; exhaling valve closed. All exhaled carbon-dioxide-laden gas is re-inhaled.



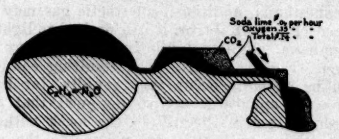
D. *Total rebreathing—later.* Carbon dioxide reaches excessive amounts; oxygen is depleted; *but the amount of anesthetic gas remains unchanged.*

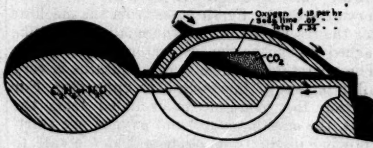


E. *Induction.* Canister removed. Accumulating carbon dioxide hastens induction. At intervals the bag is emptied and refilled with fresh gases, in order to flush out the nitrogen previously in the patient's lungs.



F. *Induction.* Canister excluded from circuit. Accumulating carbon dioxide hastens induction. Excess gas allowed to escape, in order to flush out nitrogen from patient's lungs.





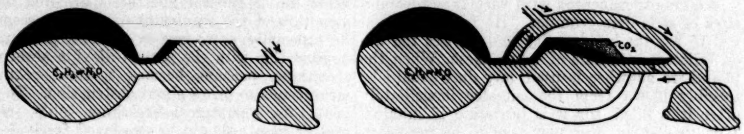
G. H. *Maintenance.* Gases pass through canister. Anesthetic gas in equilibrium with that in the blood. Oxygen used by patient compensated by slow flow from machine. Carbon dioxide absorbed by soda-lime. Notice cost per hour.

of fresh soda-lime may be slipped into place. Those who are accustomed to use it do not find it awkward to hold, for the weight of soda-lime helps to hold the mask against the face when the head is turned slightly, and the farther end of the canister rests easily in the large, soft pillow. My own experience with the to-and-fro apparatus for operations on the trunk and extremities corroborates Waters' assertion that after a few administrations one will have no difficulty with holding it in position.

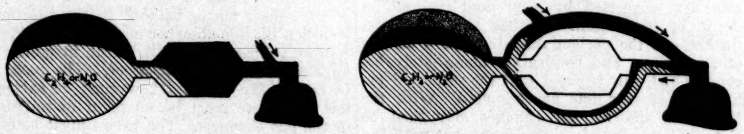
The circuit apparatus has the advantage that, when momentary stimulation of respiration is needed, the soda-lime may be switched out of and

*Guedel has reduced the bulk of portable gas apparatus nearly to the minimum. In addition to one or more soda-lime canisters, each measuring about 3 by 5 inches, he carries a "Midget" gas machine, which, when equipped with six miniature gas cylinders, fits into a carrying case that measures only 9 by 12 by 12 inches. This diminutive apparatus, with four spare miniature cylinders, is amply sufficient for a half day's anesthesias.

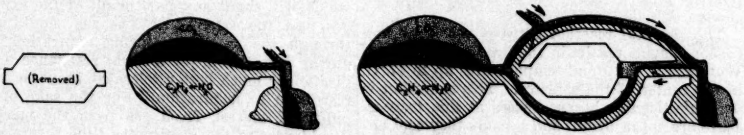
into the reservoir system with ease and without interference with the anesthetic mixture, whereas during removal and replacing of the canister of the to-and-fro apparatus the gas becomes diluted with the air which diffuses into the canister (Fig. 5: M, N). If respiration needs continual slight stimulation, the shunt valve may be opened only part way, so that any desired portion of the exhaled gases will avoid the soda-lime (Fig. 5Q). With the to-and-fro apparatus this can be accomplished only by recourse to connecting tubes of different lengths between the mask and the canister, in order to alter the amount of "dead space"[17, 18] (Fig. 5P). In operations on the head and neck the circuit apparatus is out of the way. It appeals to most anesthetists who have not used either apparatus as being less awkward to handle. Those who favor the to-and-fro method object to the circuit apparatus chiefly on the ground that the valve and tubes must offer serious resistance to respiration. At the Lahey Clinic we have not found evidence of this in using the apparatus shown in Figure 4. In fact it is theoretically an advantage of the circuit apparatus that only during expiration must the patient overcome the resistance of the soda-lime granules whereas with the to-and-fro apparatus he must both exhale and inhale through them.



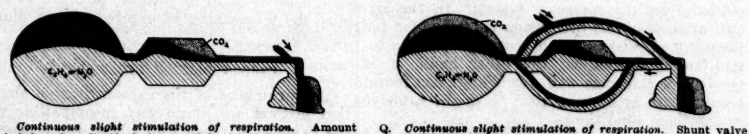
I, J. *Flooding with nitrous oxide or ethylene.* The fresh gas fills the inspiratory tube, so that the next inspiration (or more, if desired) will consist of almost pure anesthetic gas. Thus anesthesia is rapidly deepened without overcorrecting the mixture in the rest of the reservoir system.



K, L. *Flooding with oxygen.* Oxygen fills the inspiratory tube, so that patient may be oxygenated rapidly without overcorrecting the mixture in the rest of the reservoir system.



M, N. *Momentary stimulation of respiration.* Soda-lime canister excluded from reservoir system. Carbon dioxide and oxygen supplied from the gas machine.



P. *Continuous slight stimulation of respiration.* Amount of inhaled carbon dioxide is increased by lengthening the "dead space" between mask and canister.

Q. *Continuous slight stimulation of respiration.* Shunt valve is so set that part of the exhaled gas passes through soda-lime, losing its carbon dioxide, and part passes around it, keeping its carbon dioxide.

Minor points of technique. The foregoing description covers the principles involved in the carbon dioxide absorption technique and the general method of using the apparatus. The following additional practical points will be helpful in the application of this method.

Either type of apparatus may be attached to any gas machine that is capable of giving a steady flow of oxygen[3] at rates from 200 cc. per minute up (3.2 gals. per hour). Gages for measuring these unusually slow flows are fur-

nished as optional equipment on several makes of gas machine. Other machines, although able to deliver these small amounts, may not have gages capable of measuring them with the needed degree of accuracy. At small expense these can easily be equipped with an oxygen-flow meter on a shunt*, which may be brought into action only when the carbon dioxide absorption technique is used.

Waters experimented with various grades and sizes of soda-lime granules. He found it essential to use the best quality and advises the use of Wilson's soda-lime of 4 to 8 mesh. When the to-and-fro absorber is used, any dust should be forcibly blown from the canister immediately after it is filled, for it is irritating if inhaled. This precaution does not seem to be necessary with the circuit apparatus. One filling of soda-lime (500 Gm.) will last for six to ten hours of anesthesia. When it becomes exhausted, carbon dioxide accumulates. Its exhaustion is usually first suggested by a gradual increase in depth of respiration. If then the anesthetist flushes out the carbon dioxide with a stream of fresh gases and respiration immediately becomes quiet, this is proof that the suspected exhaustion of soda-lime has occurred. With the to-and-fro absorber the canister may then be changed for a freshly charged one, but with the circuit method the flow of gas must be continued to the end of the operation, unless the apparatus is so designed as to permit rapid exchange of soda-lime cartridges.

The chemical reaction between soda-lime and carbon dioxide results in the production of heat. Waters [17, 18] has found temperatures up to 47°C. in the center of the canister. This condition is not objectionable because gases at this temperature are not injurious and because they cool appreciably before reaching the patient. However, when carbon dioxide is to be added from the tank, the soda-lime canister should first be excluded from the reservoir system. In the circuit apparatus this precaution is mainly for the purposes of efficiency and economy. In the to-and-fro apparatus, on the other hand, it is needed for safety as well, for Waters has found temperature as high as 100° C. in the canister under these conditions.

It has been our experience that what would be considered a tight fit of the mask to the face for the ordinary method of giving gas is likely to allow a slow steady leak under the more severe test of the carbon dioxide absorption technique. To be leak-proof, the fit must be perfect. The use of a retaining strap passed behind the patient's head is almost essential. In case of a persistent leak, the bag must not be allowed to remain partly deflated, but the loss should be compensated by a slow flow of gas from the machine; otherwise it would be possible for the patient to draw in air beneath the mask during inhalation[4], and the resulting dilution of gases would lighten anesthesia or might bring about asphyxiation by nitrogen.

It is of the very first importance in all forms of general anesthesia to keep the respiratory passages free; yet unfortunately this precaution is frequently neglected. Boothby's statement is well worth remembering: that at least 95 per cent of all anesthetic emergencies during operation under general anesthesia are due to obstruction of the respiratory tract. It seems to be especially necessary to exercise precautions against this with the carbon dioxide absorption technique, perhaps because a tight retaining strap tends to bring about obstruction. To prevent this, pharyngeal breathing tubes (either oral or nasal) should be employed freely.

Specifications for circuit apparatus. There are on the market other styles of apparatus than those shown in the accompanying figures.* Most, if not all, of these use metal disc valves in place of the rubber flutter valves. In the selection of a carbon dioxide absorber of the circuit type the following points should be borne in mind: The apparatus must be leak-proof. The resistance to respiratory ebb and flow must be extremely low and there must be no point of narrowing in the gas channels. The valves must be of extremely easy action when wet as well as when dry, and must not permit more than a slight amount of back-flow. There should be a valve which will shunt any part or all of the expired gas past the soda lime. The gas should pass through the soda-lime during expiration, not during inspiration, for a slight resistance to expiration is much better tolerated than the same resistance to inspiration. For the sake of rapidity of control, fresh gas from the machine should pass to the inspiratory tube before mingling with the large volume of gas in the bag. "Dead space" in and adjacent to the mask should be as small as possible in order to minimize the rebreathing of expired carbon dioxide laden gas.

CARBON DIOXIDE PERCENTAGES

The principles of the carbon dioxide absorption method are in no way incompatible with the theories of those who advise the occasional addition of carbon dioxide to anesthetic gases to produce momentary stimulation of respiration or circulation. This apparatus gives the anesthetist far more complete control over the carbon dioxide. With it he can still build up carbon dioxide by excluding the soda-lime either wholly or partly from the circuit (Fig. 5: M, N, P, Q), can even add more carbon dioxide from the gas machine when indicated (Fig. 5: M, N), and yet has it also within his power to remove the carbon dioxide when he desires quiet, effort-

*The Foregger Company carries these oxygen meters.

*Apparatus shown in accompanying figures is made by The Foregger Co., New York City, manufacturers of Gwathmey and Metric gas machines.

less breathing (Fig. 5: G, H). For two reasons I cannot agree with those who advise the continuous addition of up to five per cent of carbon dioxide to the anesthetic mixture. In the first place the same high concentration of carbon dioxide can be maintained by merely reducing the flow of gases, for this would cause more rebreathing, as previously explained. In the second place it appears probably disadvantageous to maintain relatively high concentrations of carbon dioxide over considerable periods of time. The carbon dioxide content[9] of the air ordinarily inhaled is 0.04 per cent, of that in the pulmonary alveoli approximately 5.6 per cent., and of the expired air about 4.0 per cent. In mines, according to Haldane[9], the ventilation should be sufficient to keep its concentration down to one per cent, because higher concentrations, such as three per cent, incapacitate the miners for work. The inhalation of six per cent carbon dioxide causes headache, vertigo, and often unconsciousness. The advisability of reaching this and even higher concentrations by continuously adding five per cent of carbon dioxide to the fractionally rebreathed mixture, would seem very doubtful, especially in view of the advantages of lower concentrations of carbon dioxide to be described later.

Parker and McDonald[12] have analyzed the gases removed from various parts of the carbon dioxide absorption circuit apparatus (the type shown in Figure 3). With the soda-lime cut out of the circuit and fresh gases flowing at the rate of 1.5 L. per minute, they found the concentration of carbon dioxide in the inspiratory tube to vary from 1.25 to 2.00 per cent. This use of the apparatus corresponds to that of the common fractional rebreathing type of apparatus, with a very slow flow of gas. With the soda-lime in use and the anesthetic gas entirely shut off, the exhaled gases contained from 1.7 to 3.5 per cent carbon dioxide before passing through the soda-lime, and from 0.1 to 0.9 per cent after passing through the canister and while awaiting inhalation. Wienecke, Wirth, and Kirchner[19] in Germany made similar analyses, using a flow of one to three liters of fresh gas per minute. They found the carbon dioxide content of the exhaled gasses lay between 0.7 and 2.1 per cent before passing through the absorber, and was reduced to between 0.1 and 0.3 per cent after passing through. Foregger[4], using unanesthetized men, and oxygen in place of anesthetic mixtures, has found the amount of carbon dioxide in the inhaling tube always less than 0.1 per cent. Waters[17, 18] made various analyses with the to-and-fro apparatus and found that that portion of the expired gas which had passed completely through the soda-lime into the breathing bag contained not more than 0.2 per cent carbon dioxide. These analyses indicate that either type of apparatus effectively maintains a level of carbon dioxide below the point of marked physiological effect and yet safely above the zero point.

ADVANTAGES

Quiet breathing. The carbon dioxide absorption technique has been used at the Lahey Clinic for two years and has proved its great superiority. Not only has there been a marked saving in the amount of gas used, but, a matter of greater importance, the character of gas anesthesia has been greatly improved. Breathing is usually delightfully quiet, offering a marked contrast to the deep, vigorous respirations frequently seen with the common method of giving gas. Without the removal of carbon dioxide, breathing frequently resembles the panting of one who has run up a flight or two of stairs. To subject a seriously ill patient to prolonged stimulation so powerful that it calls forth this energetic, often labored, response can hardly be beneficial and might occasionally add a severe burden. The quiet breathing which results when carbon dioxide is removed bespeaks a marked saving of the patient's energy. The contrast in the general appearance of the patients is indeed striking, especially in those of marked bodily vigor or with high metabolic rate.

Moreover, the resultant quieting of the movements of the abdominal wall and contents is a boon to the surgeon when working within the abdomen. This is true whether gas anesthesia is used alone or in combination with regional or local anesthesia, or is used merely to produce unconsciousness in an apprehensive patient under spinal anesthesia. The field of usefulness of gas anesthesia is thus broadened.

Conservation of heat. Since the exhaled gases are at body temperature and are nearly saturated with moisture, the patient conserves his body heat and moisture when re-inhaling these gases, whereas if he were inhaling gases fresh from the tank with each inspiration he would be called on to warm and moisten the entire volume of gas used. It has been roughly estimated[1] that the circuit breathing method saves the patient up to 25 calories* per minute in warming the gases and 150 calories in evaporating water to moisten the gases, as compared with the method which does not use rebreathing, and that with the to-and-fro method the saving is even greater, approximately 25 and 200 calories respectively for these two items. Thus the patient's energy is further conserved. Waters[17, 18] has found that the axillary and rectal temperatures tend to rise when the to-and-fro apparatus is used. Since loss of body heat and fluid is believed to contribute to the production of shock, the decrease of this loss appears to add to the advantages of the carbon dioxide absorption method.

*Small calories, not the large calories usually referred to in medicine. (1000 small calories = 1 large calorie.)

Increase of oxygen percentage. It frequently seems that anesthesia can be maintained with a greater percentage of oxygen than it would be possible to use with the usual fractional rebreathing method. This is in agreement with Guedel's observation that the addition of small amounts of carbon dioxide to anesthetic mixtures lightens anesthesia and that their removal deepens it.

Diminution of vomiting. Vomiting after thyroid operations has been greatly reduced since we started using this method.

Decrease of explosion hazard. When the inflammable drugs ether and ethylene are used, the danger of explosion from internal static spark appears to be practically eliminated, since the interior of the reservoir system is continually drenched with moisture condensed from the exhaled gases. With ethylene, however, this advantage is partly offset by the fact that the anesthetist can never know the approximate proportion of the gases he is using (except by rapid methods of gas analysis) as he can with the usual fractional rebreathing method. He may therefore frequently be using the weaker, explosive dilutions of ethylene, which he could easily avoid by the common method of administration. Even this condition would be dangerous only in the presence of a marked leak from the apparatus, so that escaping gases could reach an external source of ignition, as for example an actual cautery or a static spark on the outside of the apparatus. With total absence of leaks the danger from external sources of ignition is entirely eliminated; therefore the method seems far safer than the fractional rebreathing method. If, however, a marked leak is present, it would seem advisable to exercise the same precautions as with the fractional rebreathing method, including the maintenance of high relative humidity in the operating room and the metallic interconnection of all parts of the gas machine.

Approach to the ideal. Ethylene given by the intratracheal route with complete rebreathing and carbon dioxide absorption, as described by Guedel and Waters[8], appears to approach nearer to the ideal than any other of our present forms of general anesthesia for operations for which deep relaxation is not essential.* The breathing is as quiet as with chloroform, yet the agent used is the least toxic of all our general anesthetics. With suitable preliminary narcosis, the gaseous mixture may contain as much oxygen as does air. It is impossible for uncontrollable respiratory obstruction to develop. The depth of anesthesia is under very quick control. Relaxation may be increased by adding small amounts of ether or by combining the method with local or regional anesthesia.

*Complete rebreathing with carbon dioxide removal is obtained in intratracheal anesthesia by the use of a large caliber catheter equipped with the small Waters-Guedel balloon which, when inflated, completely blocks off the space between the catheter and the walls of the trachea.

SUMMARY

With the carbon dioxide absorption method the same anesthetic gas is inhaled over and over again throughout the course of anesthesia, the oxygen used by the body is replaced by a slow flow of oxygen from the gas machine, and the excreted carbon dioxide is removed from the exhaled gases by soda-lime. Two types of apparatus are available to accomplish this. The expense of gas anesthesia is very greatly reduced and the danger of explosion is lessened. The patient's body heat and moisture are conserved, and his breathing is made quiet and effortless. The quieter breathing results in further conservation of the patient's energy, and renders abdominal surgery less difficult. The method appears to represent so distinct an advance in gas anesthesia as to deserve universal adoption.

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SMALLPOX AND VACCINATION*

BY SAMUEL B. WOODWARD, M.D.†

FOR seventeen years I have been puzzling my brain to find out why, if vaccination against smallpox of the children in the public schools of the state is a proper health measure, it should not be so extended that it will protect also the children in the private and parochial schools.

Such children are protected in various places, notably in the state of New York; for some inscrutable reason, however, only in cities of 50,000 and more inhabitants. The Supreme Court of that state has sent out the decision that the word "school" in the law means any school, public, private or parochial, and the city of New York supplements its provisions by vaccinating not only the pupils, but teachers, janitors, and scrub women, with the result that that great city, to which come daily thousands of outsiders by sea and by land, is freer from smallpox than any state in the United States, large or small, Massachusetts not excepted.

No state health officer could write to me as did Dr. Wynne, Commissioner of Health of New York, on October 29, 1932:—

> "It is because of our insistence on general vaccination of school children that we have practically no smallpox in this city. We have had but one death since 1912, and that was ten years ago."

Absolute protection from smallpox would require infant vaccination, and also vaccination of every individual entering the city; but it remains quite evident that complete compulsory vaccination of all school children practically precludes the occurrence of the disease. This is the case despite the fact that no country in the world, bar India and China, not even Russia, is so pest-ridden with this absolutely preventable disease as is the United States of America.

The state of Washington alone, yes, the state of Utah, with a few hundred thousand inhabitants, reports yearly more smallpox than all continental Europe, bar possibly Spain and some of the Balkan countries. Turkey has almost freed herself from the disease under the leadership of Kemal; France is now vaccinating as never before; Germany, always before the great war thoroughly protected, has again protected herself; Sweden, Norway, Belgium and Denmark know it not; and Italy, under Mussolini, has now but a modicum of what it expected to have, and did indeed have in the past; Russia has brought down its 92,000 yearly cases to 3,000; but the United States goes merrily on its way with from 30,000 to 50,000 cases reported every year. The state of Utah, where compulsory vaccination is prohibited, has among its 434,000 inhabitants almost as many cases of smallpox as Russia with its 180,000,000.

Why do people come here year after year and oppose a bill designed to cover all school children? Not because they have any special predilection for the scholars of the private and parochial schools—not at all. It is because they do not believe in vaccination. That when an epidemic broke out in Middleboro, but two of the 223 persons affected had ever been vaccinated, and those two more than 30 years before, makes no impression on their minds. That of the 60 cases of smallpox in Fitchburg last year, 57 occurred in unvaccinated persons, and that the other three had been vaccinated more than 40 years before, and not a single case in either of these in a recently vaccinated individual, passes lightly over their heads. That the one person to acquire smallpox from the gentleman who attended a dance in Williamstown while suffering from a mild attack, was the only unvaccinated person in the party, affects them not at all.

We heard here last year that there were several deaths in Fitchburg due to vaccination, and as that statement may be made again today, I would like to read the following letter:

> *"Dear Doctor:*
> Regarding your letter of February 28th regarding vaccinations in the City of Fitchburg: this is to advise you that there is no record of any deaths caused by vaccination in the City of Fitchburg."
>
> Very truly yours,
> FRED K. BRIGHAM, Agent,
> Board of Health,
> Fitchburg, Mass.

Oh, but smallpox is now a minor disease rarely fatal, is not what it used to be, and may be neglected—yes indeed! There were no deaths in Fitchburg, there were no deaths in Williamstown, there were no deaths in Middleboro—quite true; and for some years the death rate has been in general low; and so it has been before this in the past, and so it will be again in the future; but even during the years when the mortality of smallpox is low, every now and then, every here and there, it suddenly breaks out with extraordinary virulence.

"During the latter part of December, 1931, and the early months of 1932," says the provincial officer of health, "British Columbia experienced one of the most serious outbreaks of smallpox that ever occurred in this Province.

*Read before the Committee on Public Health of the Massachusetts Legislature, March 7, 1933.

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Serious, not merely by the number of cases, but because of the virulent type of the disease. There were 56 cases and 16 deaths, a mortality of 28.57%." This is recent history, surely. Mild smallpox was prevalent, and is always prevalent in the state of Washington, which has no compulsory law, and its origin was in one case at least traced to that source.

In Minneapolis in 1924 there were 994 cases and 221 deaths, and in 1925, 436 cases and 144 deaths, and from 1903 through 1931 there has never been a single year without a death in Minnesota, which during that period has had no less than 60,841 cases of the disease.

Thirty-five per cent of those affected in a recent outbreak in Oklahoma died, sixteen per cent in Tucson, Arizona, thirty-nine per cent in Denver, Colorado, forty-six per cent in Kansas City, where 123 of the 350 stricken down died. I could cite many other fatal outbreaks did time permit.

We in Massachusetts have had our experiences with the disease. Smallpox was present in epidemic form practically every ten years from the landing of the Pilgrims to the introduction of vaccination by Benjamin Waterhouse of Boston. There were nine epidemics in the first forty-seven years after settlement of Boston. We have records of outbreaks in 1690, 1702, and in 1721 when 5.759 of the 12,000 inhabitants of Boston were stricken, of whom 844 died. There were 4,000 cases and 500 deaths in 1728, and 7,669 cases with 569 deaths in 1751. Washington's army was almost paralyzed by an outbreak in 1775.

In the 180 years before vaccination was employed, there were twenty distinct general outbreaks of smallpox, with the disease always insidiously present to some extent. In the 130 years since the introduction of vaccination, there have been two, and two only, and yet there are people who say, and some of them are probably here in this room today, that vaccination is of no value, and even go so far as one gentleman who took the trouble to write to me from Louisiana to say that smallpox is not even contagious.

We protect fairly well the community in a state where education and school attendance are compulsory, by vaccinating all public school children; and I am here asking you simply to extend a law that has been on the statute books since 1855, to cover the thousands who now attend the private and parochial schools, as well as those attending schools supported directly by the taxpayers. I come here not only as an individual, but as the representative of fifty-two private schools, four State Normal Schools, and the presidents of seventeen colleges who all ask for this legislation; fortified also with the approval of the Cardinal Archbishop of Boston, and the two other Catholic bishops of the state, Feehan of Fall River, and O'Leary of Springfield. May I also say that during and preceding the agitation for this amendment the colleges in self-protection have taken the matter up, and that Smith, Wellesley, Mt. Holyoke, Radcliffe, Wheaton, Amherst and Harvard will no longer receive unvaccinated students for instruction; and for your information I may say that the same rules are in effect at Brown, Yale, Wesleyan, Cornell, Dartmouth, Vassar, Syracuse, Rutgers, Berea, Tuskegee, Hampton, Stanford, University of Pennsylvania, University of California, Alabama Polytechnic, University of Texas and the United States Army and Naval Academies.

We who come here today ask you to continue in force the most beneficial law which for eighty years has kept Massachusetts in the leading position it holds in the fight to prevent the occurrence of one of the most terrible, most filthy, most destructive of all diseases, and to extend its provisions to end the illogical stand that, while she protects a portion of the community, she allows more than 100,000 children to attend school exposed to contagion whenever smallpox breaks out in the community.

LYMPHOSARCOMA OF THE JEJUNUM

BY ERWIN H. TAYLOR, M.D.*

As sarcoma of the jejunum is not a common occurrence, the following case that recently came under my observation seems worth reporting.

Sarcoma is much less frequent in the small intestine than carcinoma, and lymphosarcoma is the usual type found in this locality.

Out of 274 autopsies on patients dying of sarcoma at the Vienna General Hospital the lesions of only three were in the intestine. Libman reported fifteen in the duodenum and eighteen in the jejunum and ileum. The majority of patients die within nine months from general cachexia or pulmonary involvement, only one case is recorded living as long as twenty-one months. The growth starts in the submucous lymph follicles, extends in the long axis of the gut and invades the muscular coat, causing paralytic distention. It may ulcerate, bleed and perforate. Sarcoma seldom invades the peritoneal coats.

Ewing in his work on neoplastic diseases says that lymphosarcoma, representing a lawless proliferation of the reticulum cells and their deriv-

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atives may be attributed to any irritant which influences these unstable cells over a considerable period of time. He also states that lymphosarcoma produces an aneurysmal dilatation of the intestine. Complications include peritonitis, obstruction of the bile ducts with jaundice and constriction of the intestine. Metastasis appears early in the regional lymph nodes and may extend to many organs.

Symptoms: The general health is impaired early, the patient rapidly becoming emaciated and anemic. There is a peculiar want of proportion between the rapid impairment of the physical condition and the absence of local symptoms. There is weakness and general debility generally with an absence of abdominal pain and no symptoms of stenosis. Occasionally constipation alternates with diarrhea. The tumor appears early and grows rapidly.

REPORT OF CASE

Mr. T. G., a barber, aged 50, was first seen on July 11, 1932, complaining of a heavy feeling across the abdomen with cramps and bloating.

He had never had any serious illness previous to the present trouble. About a year ago, he first noticed that he did not feel so well as usual, gradually losing strength and, during the last four months, losing twenty pounds in weight.

His appetite is poor, he tires very quickly and gets short of breath on slight exertion.

He is very nervous, complains of insomnia, and has considerable belching of gas and rolling in the bowels. He has some difficulty in voiding.

The bowels have required a mild laxative since he has been ill.

He uses tobacco moderately, but not alcohol. His weight was formerly 150 pounds.

On examination the patient did not look well and was considerably emaciated. The eye reflexes were normal, the tongue was coated, the teeth were in fairly good condition. The throat appeared normal, there were no enlarged glands; the pulse was regular with a rate of 120; knee jerks were present; there was no tremor; the Romberg was normal; the weight 126, the heart sounds were rather weak and rapid but there were no murmurs, and no apparent enlargement. Nothing abnormal was found in the lungs.

The abdomen was considerably distended and symmetrical, with dullness in the flanks which were tympanitic with the patient lying on the opposite side. There was considerable rumbling in the bowels. Rectal examination disclosed an enlarged prostate.

Stool was normal in color, mushy, digested, with no occult blood.

One hour after a test meal 2½ cc. of gastric contents were recovered. The total acidity was 72; free acidity 44; there was no blood.

Metabolism test was plus 28; the pulse was 120.

The urine was normal.

A gastrointestinal series had been done two months previous to the time I saw him and no pathology had been found.

The patient entered the House of Mercy Hospital and Dr. Dodd removed five quarts of bloody fluid from the abdomen. We could then feel a mass. The fluid contained mononuclear cells and a few large cells with mitotic division figures suggestive of carcinoma. Red blood cells were present. About two days later the patient was as distended as before and started to vomit, running a temperature around 101. From this time on he kept very little in his stomach and rapidly lost strength, dying on August 4.

The autopsy was performed by Dr. H. M. Scoville.

AUTOPSY REPORT 72—AUGUST 5, 1932

The body is that of a rather small emaciated white elderly male. The skin is pale and the eyes and cheeks are sunken. The abdominal cavity is distended and the ankles are swollen.

On opening the body about 500 cc. of turbid yellowish fluid are found and all the superficial loops of intestine are greatly dilated. On opening the chest nothing of note is found. On more careful examination of the abdominal cavity the most marked distention is found in the stomach and the upper portion of the jejunum while the ileum and large intestine are comparatively collapsed. About three feet from the fossa of Treitz there is an area of diffuse swelling of the jejunum, with an associated thickening of the wall and enlargement of the mesenteric nodes.

Heart: The heart is small and flabby, weighing only 185 grams. The cavities are all small and the myocardium is very thin, the right side measuring only 2 cm. and the left 8-10 cm. There is no change of note in any of the valves and the mouths of the coronaries are patent and the walls thin.

Lungs: The lungs are both voluminous and air-containing throughout. On each side there is an area of congestion and bogginess posteriorly.

Spleen: The spleen is of good size and feels firm. It weighs 130 grams and measures 10 x 6½ x 3½. It is of the usual color and consistency.

Liver: The liver is rather large, weighing 1900 grams and measuring 20 x 13 x 6½ cm. There is nothing of note externally or on section except a small very firm white nodule at the margin in the interlobar notch. On section it is firm and white

Pancreas: The pancreas is firm and white and weighs 65 grams.

Adrenals are well preserved and show nothing of note.

Kidneys: The kidneys are equal in size, weighing 185 grams each. They show nothing of note externally or on section.

Stomach and intestine: The stomach and intestine are dilated with gas down to the point of the tumor noted above. The tumor shows as a diffuse infiltration of the wall completely encircling the gut and extending for about 20 cm. The wall of the intestine in this area is very thick and on section is white and cellular. The mucosal surface is red and shows a surface membrane which is a dirty grayish white in color. The lymph nodes in this portion of the mesentery are very large and form an irregular firm mass about 8 x 10 cm. On section they appear white and cellular and very similar to the tumor seen in the wall of the intestine. There are a few retroperitoneal nodes found which are smaller but white and evidently involved with the tumor. These are quite close to this same area so that there is no widespread involvement of the lymph nodes.

Thyroid: A small portion of the thyroid is removed and appears yellowish brown in color with minute beads on section, probably due to colloid.

AUTOPSY REPORT 72—MICROSCOPIC NOTES

Heart: The section of the heart shows the fibers to be small with small nuclei. There is a yellowish shadow suggestive of brown atrophy. Otherwise the section shows nothing of note.

Lungs: On section, the lungs show delicate walls and empty alveoli except for an occasional endothelial cell packed with old brown pigment. Another section taken through the posterior portion of the lungs shows the alveolar spaces filled with fluid and numerous pigment carrying cells. In some areas the process has progressed and red cells and a few polymorphonuclear leukocytes are seen. There is a tendency for the alveolar walls to break down.

Liver: The sections of the liver tissue show no change of note. The sections taken through the small white nodules noted grossly show it to be made up of dense partially hyalinized connective tissue with large thick-walled blood vessels running through. A few small islands of liver tissue are seen and a few collections of round cells.

Kidneys: The sections of the kidney show nothing of special note. There is some precipitated serum in the tubules and the lining cells are swollen and a little irregular but there is no cellular reaction.

Intestine: The sections taken through the tumor noted grossly in the jejunum all show essentially the same picture. All the coats of the wall are replaced by tumor tissue which is made up of round cells of varying sizes, all of which show an abundant cytoplasm. These cells lie in sheets with no very characteristic architecture, many of them showing mitotic division figures. There are a few small round cells seen scattered among the larger cells which make up the bulk of the tumor.

Lymph nodes: The sections of the lymph nodes of the mesentery show dense infiltration with the same type of cells as those seen in the intestine.

Thyroid: The sections of the thyroid show nothing of note.

Large cell lymphosarcoma of the jejunum with metastases in the mesenteric nodes: ascites, dilatation of the upper jejunum and stomach.

Atrophy of the heart, hyperstatic pneumonia.

Fibroma of the liver.

RUPTURED ANEURYSM OF THE HEPATIC ARTERY

BY ERWIN H. TAYLOR, M.D.*

THERE seems to be very little in medical literature concerning the rare condition of ruptured aneurysm of the hepatic artery; in Keen's Surgery it is not even mentioned and according to Tyson's Practice of Medicine it is a rare lesion and only ten or twelve cases have been reported.

Blumer states, "Aneurysm of the hepatic artery is rare, occurring once in 500 cases of aneurysm in general. It may be either a true or false aneurysm. The latter is met with chiefly in conjunction with a gall stone which has become impacted in the cystic or common duct and has led to erosion of the outer coats of the artery. In that very rare disease, periarteritis nodosa, the liver is often involved showing many small aneurysms.

"Death takes place usually from rupture of the aneurysm either into the peritoneal cavity or into the bile passages. Repeated hemorrhages over a period of several weeks have been recorded.

"Pain is nearly always the most prominent feature—it is situated in the epigastrium and right hypochondrium and is either constant or more frequently intermittent and resembling biliary colic. A palpable tumor is almost never present and visible pulsation has not been recorded.

"The diagnosis is seldom possible during life. The combination of biliary colic and repeated attacks of gastro-intestinal hemorrhage are suggestive of hepatic aneurysm, but occur also and more frequently in connection with cholelithiasis."

Mr. A. S. aged 22, single, entered the hospital January 3, 1932, suffering from a severe hemorrhage from the stomach, and acute pain in the epigastrium. There was a history of some pain and distress in the epigastrium about a year before. Four months before admission he had an attack of acute pain in the right lower quadrant followed by the removal of a gangrenous appendix. Otherwise his previous history was not important.

Father died of alcoholism. Mother living, in good health. Two brothers died as a result of hemorrhages from the stomach; one sister living and well.

On admission the patient, a tall slender boy, was pale, weak, nauseated and was vomiting blood. The pulse was 112 but regular, and of fair quality. The pain in the epigastrium was severe. He was put to bed with an icebag over the epigastrium and given nothing by mouth and morphine administered hypodermically. Coagulose was also used. Fluids were administered, introduced by rectum and intravenously.

The main points on the general physical examination were the anemia and epigastric tenderness.

The Wassermann was negative.

The urine was normal.

Stools were black and contained blood.

Blood showed hemoglobin 55%—R.B.C. 3,616,000—W.B.C. 6,300—Polys 63%—Small Lymphocytes 34%—Large Mononuclears 1%—Eosinophiles 1%—Blood platelets 272,000.

Bleeding time was 5 minutes.

Coagulation time was 3½ minutes.

After five days of starvation, liquids and light nourishment were started by mouth. About ten days following the hemorrhage he had an attack of hives which lasted for several days, probably caused by the coagulose. During this attack he vomited some, but without blood. After the vomiting had subsided he was given a gastrointestinal series. The pictures showed a poorly filled duodenal cap and an empty stomach at the end of six hours. The intestinal picture showed nothing abnormal.

Two days after the x-rays were taken, he was seized with pain and vomited much blood, continuing to do so for two or three days. During this time he was given a blood transfusion. From this time on he improved on a careful Sippy regimen and was discharged from the hospital February 6, free from pain, gaining weight, and with color much improved, but there was still a positive occult blood test in the stool.

He was kept in bed at home for a month longer and then allowed to get up and walk about. He had no pain, and took his diet well until March 14 when he

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was seized suddenly with an attack of severe pain in the epigastrium and vomited a lot of fresh blood. Temperature was 101 and pulse was 104.

On May 18 the patient's condition had improved sufficiently so that an operation seemed advisable.

The duodenum was found adherent to the gall bladder and the pancreas was enlarged and indurated. There was a hard mass about the size of a walnut situated apparently in the posterior wall of the duodenum.

A posterior gastro-enterostomy was done and the patient made an uneventful recovery.

He was kept on a carefully selected diet and gained in weight and strength and did very well until about the middle of June when he again complained of acute pains in the epigastrium with some nausea. On June 25 he had another attack of pain and vomited a lot of blood. He was sent to the hospital and given a blood transfusion and the usual treatment for hemorrhage from the stomach.

His condition improved very slowly and he had more or less pain and nausea nearly every day. He was a poor surgical risk but it was felt that nothing but surgery offered any hope so on July 29 he was again operated upon. There were very dense adhesions and a mass was found about the size of an ostrich egg apparently involving the posterior part of the duodenum. This mass was much larger than at the previous operation. There were many enlarged retroperitoneal lymph glands. As it was impossible to remove the mass, the wound was closed. The patient had a very stormy postoperative period and finally died on August 13.

Autopsy performed three hours postmortem by Dr. R. S. Woodruff and Dr. Forsyth.

General Condition. The body is that of a fairly well-developed but markedly emaciated white male, appearing to be of the stated age. The skin has a normal texture but is of an icteroid tint. There is practically no subcutaneous fat present. The pupils are equal and measure about 0.5 cm. The teeth are in good condition and there are no enlarged lymph glands present to be palpated externally.

Thoracic Cavity. The heart and lungs bear a normal relationship. On opening the thorax, there was an outpouring of gas from the left pleural cavity and the left lung was found collapsed. On the right side the pleural cavity was obliterated throughout by dense, fibrous adhesions, holding the lung firmly fixed. There was no fluid in either side.

Pericardial Cavity. Contained about 100 cc. of clear, bile-stained fluid. No adhesions present.

Peritoneal Cavity. There is a linear scar present to the right of the mid-line about 12 cm. long, following the rectus muscle. There are also several small, discharging sinuses in the abdominal wall about the incision. The omentum and a portion of the anterior wall of the stomach are firmly bound down to the incision area by thick adhesions. There are numerous adhesions binding together the coils of small intestine and also many about the liver and gall-bladder. The gall-bladder is greatly distended and fluctuant. Posterior to the gall bladder and along the right paracolic gutter, a large abscess cavity was opened into, from which drained about a pint of thick, fecal smelling purulent material. The posterior surface of the stomach and the lesser curvature are firmly bound to the liver by many dense fibrous adhesions. Behind the pyloric end of the stomach and the first part of the duodenum may be felt a firm, rounded, slightly nodular mass which appears to be a thick, fibrinous laminated blood clot, about 6 cms. in diameter. The sac in which the clot is contained is composed of thick adhesions and also a dilated portion of the hepatic artery which has ruptured. The aneurysmal sac measures about 3 cm. in length and 2 cm. in width. The walls of the sac are thin and rather friable. There is also a small erosion into the portal vein. A portion of the blood tumor has undergone softening and has become infected, lodging a small amount of thick purulent material. This area communicates with the erosion into the portal vein. The sac has also disintegrated the head of the pancreas for a distance of about 1 cm. The remainder of the sac is firmly attached to the pyloric end of the stomach and the first part of the duodenum, but there is no evidence of perforation into either.

The liver is large and firm, but presents on the upper surface many small nodules which are soft and fluctuant, and which appear to be filled with fluid. The liver is of a mottled greenish and purple color. The gall bladder is large and distended. The common duct is apparently not patent as no bile can be expressed. On opening the gall bladder it is seen to be filled with a thick, greenish, purulent material of a distinctly fecal odor.

The retroperitoneal glands are much enlarged, soft, and of a dark, greenish color.

The intestines appear to be normal exteriorly, and there are no pelvic abnormalities.

Heart: Slightly smaller than normal, soft, and of a deep reddish-brown color. Small amount of pericardial fat. The heart was left in situ and the chambers not opened.

Lungs: The right lung is rather small, soft and doughy, and of a light pinkish color. The pleural surface is dull and covered with numerous tabs of fine, fibrous adhesions. At the apex there is a small scarred area about 1 cm. in width. The cut surface of the upper lobe is dry, granular, subcrepitant throughout and of a pale yellowish color. The middle lobe is similar. The lower lobe is crepitant though rather soft and doughy. The surface is spongy, of a deep pinkish color, and the base shows some passive congestion. A small amount of bloody fluid may be expressed. The left lung is very small, collapsed, subcrepitant throughout, and very doughy in consistency. It is of a pale pinkish yellow color throughout. The cut surface of the upper lobe is dry, granular, collapsed and light yellow in color. The lower lobe is similar.

Liver: The organ is large, firm, smooth on the surface except for numerous small nodules scattered throughout the whole surface, most marked on the upper diaphragmatic surface. The whole liver is mottled green and purple in color. The cut surface is of a dull, brownish-green color, and there are numerous small abscesses scattered throughout the substance filled with thick, greenish, purulent material. The majority of the abscesses are about ½ cm. in diameter. Some of the portal vein branches show wasting and toxic degeneration.

The gall bladder is much distended and filled with purulent material and the mucous membrane is degenerated.

Spleen: The organ is moderately enlarged, firm, smooth surfaced and of a deep purplish color. The capsule strips fairly easily. The cut surface is fairly firm, moist, mottled purple and yellow, the germinal centres being pale. It has a definite sago appearance and shows evidence of chronic wasting and toxic degeneration.

Esophagus: No gross lesions.

Stomach: The organ is of a normal size and shape, smooth and shiny on the external surface and of a light grayish color. The wall is of a normal thickness throughout. The mucosa is rather thin, pale for the most part but with a few areas of congestion. The rugae are not very prominent and the surface is covered with thin slime and presents the appear-

ance of a mild catarrh. There is no evidence of any ulceration or past or recent perforation. The gastro-enterostomy opening is patent and firm. The pylorus is also patent and shows no abnormalities.

Intestines: The duodenum is of a normal size and appearance. The mucosa in the first portion is smoothed out where the blood tumor protrudes into it but there is no evidence of any ulceration or previous perforation. The remainder of the duodenum is negative, as are the rest of the intestines.

Pancreas: The organ is of a normal size and shape and quite firm. The head is eroded where the aneurysmal sac has eaten into it.

Adrenals: No gross lesions.

Kidneys: The right kidney is of a normal size and shape, firm and tense. It is of a reddish brown color and smooth and shiny on the exterior surface. The capsule strips easily and shows no thickening. The cut surface is moist and glistening, granular, and considerably congested. The cortex and medulla are of a normal thickness but the pyramids of the medulla are yellowish white and rather waxy in appearance. The pelvis is normal. The left kidney is similar to the right.

Bladder: No gross lesions.

Genitalia: No gross lesions.

Aorta: Of rather small diameter. The intimal surface is smooth and shiny, though about the arch there are signs of early atheroma.

Summary:

1. Marked emaciation.
2. Moderate jaundice.
3. Healed right-sided pleurisy with old healed scar in the right apex.
4. Subacute, purulent peritonitis.
5. Inflammation of the gall bladder with abscess formation.
6. Aneurysm of the hepatic artery with rupture into the peritoneal cavity; walled off by adhesions.
7. Multiple abscesses of the liver.
8. Passive congestion of the spleen.
9. Erosion of the head of the pancreas by the aneurysm.
10. Toxic nephritis.

Note: There is no evidence of malignancy in any part of the body, nor is there evidence of ulceration in the stomach or duodenum, nor any way, macroscopically, by which bleeding could have occurred into the stomach. It is possible because of the fact that the aneurysmal sac communicated with the gall bladder that the bleeding occurred into this organ and from there was transmitted to the duodenum and so to the stomach.

I am indebted to Dr. Harry Bard, Pittsfield, Massachusetts for permission to report this case.

SOME RECENT RESEARCHES IN EPILEPSY AT THE MONSON STATE HOSPITAL*

BY RILEY H. GUTHRIE, M.D.†

I SHALL summarize briefly the results of some of our recent research activities. During the past decade numerous articles which were prepared by our staff on the pathology and clinical features of epilepsy have appeared in various medical periodicals. We have approached the problem of epilepsy from several different angles in order that we might be better able to consider the whole individual in the study of his particular problem rather than to think in terms of neurone function alone or in terms of morbid anatomy of motor pathways, cerebellar tracts, basal ganglia, etc. Surely, structural changes may occur in all these without causing convulsions and conversely convulsions may occur without demonstrable permanent changes in any organ. Since epileptic patients have in common one symptom, the convulsion, which is so similar in every case that the condition is described by a single word—"epilepsy"—one naturally looks for some common etiological factor to explain the recurrence of this dramatic phenomenon. In searching for the cause of convulsions, one looks for a more simple mechanism than in looking for the etiology of disease, for the seizure must be due to a phenomenon that may occur in many pathologic states. Admitting that the predisposition or the vulnerability to epilepsy may come from many causes varying from poor inheritance to cerebral trauma, yet one looks for a common precipitating factor which touches off the explosion at intervals after the bomb has been properly prepared. With this in mind, we have approached the problem of epilepsy from a standpoint of pathological physiology. We were encouraged in this by favorable reports from therapeutic research projects, especially those which depend, for their beneficial effect, on certain physicochemical body changes[1]. I refer especially to acidosis induced by the ketogenic diet[2] and dehydration by fluid intake limitation[3]. These two therapeutic procedures have become recognized generally as valuable aids in alleviating symptoms but one may doubt if they ever result in permanent cure. There have appeared two schools of thought on this subject; one believing that the ketogenic diet relieved epileptic patients by inducing an acidosis or ketosis, or indirectly by increasing the amount of calcium ion, and the other believing dehydration relieved these patients because lessening the fluid constituents of nerve tissue raised the convulsion threshold. Since both these methods of treatment have some factors in common, i.e., loss of body fluids, shift in acid base equilibrium toward the acid side, and loss of weight, many physicians have used a combination of both methods with good results[4] and, probably, with less inconvenience to their pa-

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tients. In our study of the influence of acute infectious diseases[5] and of debilitating diseases on the course of epilepsy, we have found that a decrease in the frequency of seizures occurred in most cases. Also, we have shown that the incidence of epilepsy is low in diseases in which there is present a low blood volume or a tendency to dehydration, e.g., diabetes mellitus, cretinism, and thromboangiitis obliterans[6].

Believing that if acidosis causes a negative water balance which is associated with clinical improvement, and if strict limitation of fluid intake brings about similar clinical results, there might be a detectable decrease in the amount of circulating body fluids as a result of these therapeutic procedures, and believing that persons subject to epilepsy might show an increase in fluid constituents, we undertook a study in the blood volume of epileptic patients. Under the direction of Dr. Morgan B. Hodskins and in collaboration with Dr. James Z. Naurison, whose understanding of normal and abnormal physiology has been invaluable, we have conducted intensive research, a part of which has already been published.

The blood volume values of 78 untreated epileptic patients in interim were compared with those of 42 normal controls[6]. It was found that these epileptic patients showed an average increase in blood volume of 8 per cent over the controls, but the most striking finding was the wide range of blood volume values in patients as compared with the relatively narrow range of values in normal subjects. These findings justify the opinion that the individual who is subject to epilepsy tends to retain fluids and corroborate recent evidence that the acute convulsive state is associated with disturbed water metabolism and acute retention of body fluids[7].

Along with our studies in dehydration, acidosis, and blood volume, the therapeutic and physiologic effect of ammonium nitrate as a dehydrating agent was also studied[6]. Obviously a drug which induces a negative water balance—a drug which would act directly opposite to pitressin[8] which causes retention of water and induces seizures—would be quite useful. The blood volume in fifty epileptic patients was studied before and after the administration of ammonium nitrate 90 gr. (6 Gm.) daily. The duration of treatment varied from nine to three hundred and eighty-four days with an average treatment period of two hundred and eleven days. No strict limitation of water intake was undertaken. Treatment with this acid-forming salt caused an average decrease in blood volume of 8 per cent and an average decrease in CO_2 combining power of the plasma of 15 per cent. However, there was no clinical improvement noticed in most cases. These results show that moderate acidosis induced by administration of ammonium nitrate does not have a favorable influence on the clinical course of epilepsy. The average decrease in blood volume while under treatment was only 8 per cent and the average weight of patients remained unchanged—apologies for our inability to control strictly and limit the fluid intake of these patients. Since there was no loss of weight in this group of patients one reasonably assumes that dehydration did not occur.

It will be of interest to the internist to know that there were no ill effects from administration of ammonium nitrate 90 gr. (6 Gm.) daily over a long period of time. The non-protein nitrogen of the blood and the plasma chlorides were not materially changed after long-continued treatment.

An hypothesis which is quite generally accepted is that edema, anoxemia, and alkalosis favor the convulsive state and that dehydration, increased oxygen tension, and acidosis tend to inhibit convulsions[9]. Since cancer is thought to be associated with alkalosis in most cases[10], we reviewed the case records of 55 patients in whom cancer had complicated and modified the course of epilepsy. It was found that the number of convulsions decreased in most cases as the malignancy progressed[11]. If malignancy is generally associated with relative alkalosis, then the shift in acid base equilibrium does not play an important rôle in the face of dehydration which presumably occurred in these cancer cases. In our experience, loss of weight, whether due to disease or to restricted diet, has generally been accompanied by clinical improvement in epileptic patients. One may speculate as to whether this improvement is due to dehydration or increased oxygen tension, both of which may accompany loss of weight, but the results of our recent research tend to discredit acidosis alone as an important factor. Our experience with caffeine and diuretin with careful control of fluid intake substantiates our belief that dehydration plays a very important rôle.

Intracranial vascular conditions, upon which depends the supply of nutrient to the nerve cells, are favorably influenced by a number of drugs, among which should be mentioned caffeine and amyl nitrite and many sedatives, especially phenobarbital (luminal) and iso-amyl-ethyl barbiturate[12] (amytal). In the treatment of status epilepticus many different measures have been employed. During the past two years we have used sodium iso-amyl-ethyl-barbiturate (sodium amytal) 7.5 gr. (0.5 Gm.) intravenously in 14 cases of status with encouraging results. Sodium phenobarbital (sodium luminal) in 4 to 8 gr. doses intravenously has been very useful in the treatment of status and the margin of safety in its administration is much greater than is that of sodium amytal; however, the latter has been much more effective and spectacular in relieving the patients. The administration intravenously of sodium amytal calls for caution and should be done slowly; however, our

results have been uniformly good. The beneficial effects of these sedative drugs may possibly be explained by their vasodilator action on the cerebral vessels which in turn increases the supply of oxygen to the brain. Nerve tissue is most sensitive to oxygen want[13, 14, 22] and when the circulation is slowed and oxygen supply is limited, compensatory cerebral vasoconstriction might cause anoxemia enough to precipitate a seizure.

The relation of the acute convulsive state to intracranial hydraulics, to cerebral vasomotor control, to oxygen utilization, and to capillary permeability[15] is most intriguing. Anoxemia increases the permeability of capillaries to plasma proteins and permits water to pass through much faster than under normal conditions[16, 17]. This increase in capillary permeability may cause cerebral edema which has been frequently described in association with epilepsy. The interrelation of all these factors and the manner in which they are influenced by dehydration, acidosis, and increased oxygen tension lead one into the involved realm of physical chemistry and bio-chemistry[18]. On changing from recumbent to standing position for thirty minutes, approximately 11% of the plasma filters from the circulation into the interstitial tissues[19]. Since disturbed water metabolism is concomitant and apparently a causative factor in the acute convulsive state[20], one might expect that this filtration rate would be abnormal in epileptic patients, especially at or about the time of the convulsion. We have attempted to study the capillary permeability in epileptic patients and in normal subjects by determining their ability to concentrate blood plasma while standing. When patients were tested in interim there was no deviation from the results obtained in controls; however, two patients who had mild epileptic symptoms, in the form of petit mal, during the standing period showed no tendency to concentrate plasma by specific gravity determinations. Acute fluid retention which is said to precede the acute convulsive state may have its origin in a sudden change in capillary permeability, or possibly, a change in the colloid osmotic pressure.

Without envisaging epilepsy in terms of biochemistry, one may see in it a plausible approach to the study of individual predisposition, and of precipitating life situations which are intimately associated with metabolic changes. One is reminded of other equally dramatic conditions which are associated with autonomic phenomena, namely, vasovagal attacks of Gowers, neurocirculatory asthenia, and vagotonic crises[21]. In all these conditions and in some cases of epilepsy, the emotional element and its counterpart, the vasomotor and physiologic imbalance, are obvious, and by considering all these factors one is able to clear the barrier of the "organic" and the "functional". It may not be so important to understand the disease which the patient has, and make an elaborate differential diagnosis, as it is to understand the patient and his physiologic equipment and stability. This removes one from the primitive state of medicine, i.e., description and classification, into more modern methods where one attempts to understand physiologic mechanisms[22], giving hope of eventually determining the cause of convulsions.

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.
F. M. PAINTER, A.B., ASSISTANT EDITOR

CASE 19121

SUBSTERNAL PAIN FOR ONE DAY

MEDICAL DEPARTMENT

A Canadian restaurant cashier and waiter sixty-one years old was sent in through the Emergency Ward complaining of substernal pain of one day's duration.

Three weeks before entry while working he had intense numb constant pain originating deep under the midsternum. In the course of an hour this increased in intensity and radiated straight through to the back and down the right arm to the fingertips. It was somewhat aggravated by exertion. When he lay perfectly still it did not keep him from sleeping. The pain lasted for about two days. During the next three weeks he had two more similar attacks, the last a little more severe, keeping him in bed for twenty-four hours. Between the attacks he did not feel weak and worked as usual. The day before entry while going home from work he again had the pain. This time it persisted even when he was quiet. The character and distribution were as before. His physician gave him several doses of morphine during the next twenty-four hours with partial relief. At the time of entry the pain was still present but was gradually wearing away.

Three weeks before entry he came to the Outpatient Department complaining of epigastric pain and distention. The heart was possibly slightly enlarged. The sounds were distant. There was tenderness in the midepigastrium. An electrocardiogram five days before entry showed ventricular premature beats, rate 80, inverted T_1, coronary type, low voltage QRS. A gastrointestinal series, including a barium enema, showed only a redundant colon. A Hinton test was negative. The blood pressure was 154/110.

His family history, so far as he knew it, is not significant.

He had always been strong and healthy until recently. He had gonorrhea thirty years before entry. For eight years he had had pain in the right lower quadrant at intervals, for which he came to the Outpatient Department seven months before entry. Examination and x-rays at another Boston hospital four years previously had been negative. The Outpatient studies also failed to reveal the cause of the pain. The heart was negative. The blood pressure was 180/110.

Physical examination showed a well nourished, drowsy man lying comfortably in bed. (He had recently had morphine.) The face, lips and mucous membranes showed a red cyanosis. The heart was enlarged to the left (left border of dullness 11.5 centimeters, midclavicular line 9, supracardiac dullness 7.5). The rhythm was irregular because of frequent ventricular extrasystoles which did not come through to the wrist. The sounds were of very poor quality, faint and distant, scarcely audible even when he sat upright and held his breath in forced expiration. The artery walls were palpable. The blood pressure was 115/90 to 60/35. An electrocardiogram showed normal rhythm, rate 100, late inversion T_1, coronary type, low voltage (some overshooting because of high resistance). There was the slightest suggestion of a high origin of the T wave (Pardee's sign) as compared with the previous Outpatient record. There were medium and fine moist râles at the bases of both lungs in the back. The pupils were equal and regular but did not react to light. The reflexes were normal.

The amount of urine is not recorded. The specific gravity was 1.016 to 1.022. There was a very slight trace to a slight trace of albumin at all of three examinations. The first sediment specimen was loaded with white cells. The other two showed 2 to 4 per field. Two specimens showed red cells. All showed granular casts. Blood examination showed 6,590,000 red cells, hemoglobin 100 per cent, 14,500 to 27,850 white cells, 83 to 88 per cent polynuclears. One smear showed more than the normal number of large red cells. Another showed the red cells normal, but several band cells among the white cells. A Hinton test was negative. Stool examination was negative.

The temperature was 97° to 100.5° with a terminal rise to 103°. The pulse was 75 to 100 with a terminal rise to 120. The respirations were 12 to 30.

The patient was given absolute rest in bed and was very comfortable. The day after entry a pericardial friction rub was heard, which had disappeared by the fifth day. The number of basal râles increased. On the fifth night he complained of gradual onset of pain in the left leg just above the ankle, becoming worse during the night. The next day the left foot and lower leg felt cooler than the right and the left posterior tibial and dorsalis pedis arteries could not be palpated. The following day the popliteal could not be felt.

On the eighth morning a surgical consultant reported that the only possible chance of saving the foot was by immediate operation, which was done. The following night the foot was still cold. The second day after operation the pa-

tient appeared moribund. The foot and lower leg were gangrenous, with a good line of demarcation. The blood pressure fell to 60/35 and the temperature rose to 103°. The following morning he was unresponsive. That night his blood pressure was 80/52. He did not regain consciousness and died apparently in his sleep the following morning.

Clinical Discussion

Dr. Richard C. Cabot: With this history we should ordinarily say it is an infarcted heart. We expect pain down the left arm, but it does go to the right sometimes too.

The striking thing is that he was able to work and not prevented from sleeping in the first attack.

The electrocardiogram is perfectly in harmony with the diagnosis that we have suspected.

It is noticeable how much more the systolic blood pressure varies than the diastolic. That is the rule. The diastolic is very much more important. It is very much harder to measure. Many men do not know how to measure it and so they skip it, measuring only the systolic, though it is so much less important. I would call attention particularly to this blood pressure. You are going to be called to many cases of heart disease just at this stage and you are going to find the blood pressure low, and if you are wise you are not going to exclude hypertension as the cause of the illness, because the high pressure may have been high but may have gone down. If the case otherwise will fit hypertension, do not exclude it because the pressure is low after compensation has failed.

Dr. Tracy B. Mallory: It may also stay low, even if he recovers pretty good compensation, may it not, Dr. Cabot?

Dr. Cabot: There is a stage when a person is beginning to have hypertension when it goes up and down at all sorts of intervals. But I think that when high pressure has once become continuous so that we are sure of the diagnosis, it does not go down for any reason whatever except approaching death.

The pupils did not react to light, which probably, so near death, is not of any importance; otherwise we should have paid more attention to it.

The large number of red cells was undoubtedly due to cyanosis, I think. Band cells are undeveloped polynuclears, but I do not think they have any significance here.

A pericardial rub is a thing we are always looking for in this disease. It is perfectly conceivable that it is there, also perfectly conceivable that it is not.

The essential thing we have here is arterial embolus in one or more of the coronary arteries and in the arteries of the leg, and it may have got up to the abdominal aorta for all I know. I am curious about the operation. I do not know what it was.

Dr. Robert R. Linton: At the time I saw this patient it appeared that he had had an embolus which was lodged in the popliteal artery of the left leg. The onset of the pain in his leg, which was described as being gradual in onset, was not typical of an arterial embolus. Pain due to sudden occlusion of an artery from within is usually very sudden and excruciating. It is possible that the type of pain in this case was masked by the sedatives he was receiving for his cardiac condition. I made the diagnosis of embolism of the left popliteal artery chiefly on the physical findings. The left foot and ankle were cold, cyanotic and beginning to take on a cadaveric appearance. The left lower leg up to a few inches below the knee was much cooler than the leg above the knee. The only arterial pulsation in the left leg that could be palpated was that of the femoral artery. This artery could be traced down about two thirds of its course in Hunter's canal, where the pulsation abruptly ceased.

I assumed that the embolus had occurred sixty hours previous to the time I first saw the patient. This relatively long period probably meant that the chances of a successful embolectomy were not very great. It was however so evident what the outcome was going to be, at least the loss of the left foot, that it seemed justifiable to attempt to reëstablish the circulation.

The operation was a simple one. The popliteal artery was exposed, using local anesthesia. The embolus was located about the middle of the popliteal artery at the point where the inferior lateral and medial genicular arteries arise. A small vertical incision one centimeter in length was made over the embolus, which was located by the firmness of the clot and a slight bluing of the arterial wall. Also the artery distal was much smaller in diameter than it was proximal to this point. The embolus was milked out gently. It measured about 15 by 8 millimeters. Attached to the distal end of it there was a thin propagated clot about three centimeters long and two to three millimeters in diameter. The propagated clot proximal to the embolus was milked out. Following this procedure there was a gush of bright arterial blood. The posterior tibial artery was then exposed above the medial malleolus. It contained no blood or blood clot. It was cannulated with a ureteral catheter and washed upward with one per cent sodium citrate. When the solution came out clear from the popliteal artery, the incision in the latter was closed with fine oiled silk. On release of the popliteal artery blood was seen to come out of the posterior tibial artery, so it was sutured in a similar manner. At the time of operation it was noticed that the intima showed considerable damage at the point where

the embolus had lodged, so that I was afraid that a new clot would form at that point.

The lesson this case has taught me is that embolectomy in a peripheral artery is a relatively simple technical procedure and it should be attempted as soon as the diagnosis is made. If too long a period of time elapses before operation is attempted the chances of failure are great, due to damage to the arterial wall at the point of occlusion with resulting secondary thrombosis of the artery. At present it is not possible to say what this period of time is after which it is useless to attempt an embolectomy. Until we determine this I think one is justified in performing the operation in a case where there is a possible chance of a successful result.

DR. CABOT: We shall find an enlarged heart because of his hypertension; very possibly the type of renal changes we have talked of so often which go with perfectly good kidney function or without it. The coronaries should be plugged by a recent thrombus whether they are narrowed or not, and there is every reason to suppose they are narrowed. The origin of that leg thrombus we do not know. It seems to be a mural thrombus in the inside of the heart, from which a piece broke off and went down to the leg artery. It is very possible there was a pericarditis, which often comes on the outside of the infarcted area just as the thrombus comes on the inside of it. The main cause of death it seems to me there is no doubt of at all,—coronary thrombosis.

A STUDENT: We have been told that when the blood pressure falls we should give treatment to build it up. Is this a case where that would be contraindicated?

DR. CABOT: If it falls from shock or hemorrhage we should try to raise it. When it falls from cardiac causes we cannot build it up ordinarily. Digitalis does not do it. I do not know any drug or other treatment that does except very temporarily. We have then these two types: blood pressure falling from shock or hemorrhage, as in wounds, for which we can do a great deal, and this blood pressure falling from cardiac causes, for which I do not know any relief.

A STUDENT: Do you expect the gonorrhea had any significance in this illness?

DR. CABOT: I should say not.

A STUDENT: Could this be polycythemia?

DR. CABOT: I should say not. We do not make that diagnosis unless we can exclude congestion of the blood at the surface from circulatory causes. Polycythemia vera is contrasted with what is not *vera*, that is, simulated. This is simulated, I should say, by cyanosis.

A STUDENT: Can you get a white count as high as that from pericarditis?

DR. CABOT: I think so; but I do not think there is any reason to consider it. We can have it from pericarditis or from these thrombi alone, because I think the foreign proteins in the blood, which we must have with these thrombi, are enough to cause leukocytosis.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Coronary thrombosis.
Embolism of the left leg.
Gangrene of the left leg.

DR. RICHARD C. CABOT'S DIAGNOSES

Coronary thrombosis.
Cardiac infarction.
Thrombosis of a large artery in the leg.

ANATOMIC DIAGNOSES

Coronary thrombosis.
Hypertrophy of the heart.
Arteriosclerosis of the aorta, marked.
Embolus to the left popliteal artery.
Infarcts of the spleen and liver.
Chronic vascular nephritis.

PATHOLOGIC DISCUSSION

DR. MALLORY: We found a considerably enlarged heart weighing 500 grams, and an area of infarction at the apex of the left ventricle with a definite thrombus in the descending branch of the left coronary about one and a half centimeters from its orifice, the most frequent spot of all for coronary thrombosis. I think that is of some interest in relation to the radiation of the pain down the right arm instead of the left. The site of the infarct is also the usual one.

He had a very extreme degree of arteriosclerosis in the aorta, more particularly in the abdominal portion, with great atheromatous masses, large ulcers, and secondary thrombi on top of his ulcers. Some of the thrombi were pedunculated and measured from one half to one centimeter in diameter. So that he had two possible sources for an embolus to his leg. It might have come either from the intracardiac thrombus secondary to the infarct or it might perfectly well have come from these shaggy thrombi on the atheromatous ulcers. There is still a third possibility, that it was not an embolus but a local thrombus formation directly in the popliteal artery itself. But the vessel in that region did not show any marked degree of arteriosclerosis, so we felt that on the whole it had come from higher up, either from the aorta or from the cardiac thrombus.

The kidneys showed well marked chronic vascular nephritis. There were a couple of fairly long standing infarcts in the spleen and a quite fresh infarct in the liver,—a rather unusual finding.

DR. CABOT: No pericarditis?

DR. MALLORY: No.

DR. CABOT: If you think I get extreme on the subject of the impossibility of diagnosing pericarditis, remember this case and some of the others we have had this year.

CASE 19122

GASTRO-INTESTINAL BLEEDING

MEDICAL DEPARTMENT

PRESENTATION OF CASE

DR. ALFRED KRANES:* This is the case of a sixty year old white married Irish night watchman who was brought into the Emergency Ward one night complaining of weakness of two days' duration.

Seventeen years before entry he had gnawing epigastric pain coming on two to four hours after meals and relieved somewhat by food and soda. He had stools of dark color and was excessively weak. At the end of a year he went to a hospital, where x-ray examination showed an irregularity of the duodenum. On exploration a large mass of adhesions was found stretching from the surface of the pancreas across the duodenum and up to the under surface of the liver. The gall bladder, duodenum and stomach were apparently normal. The operation done consisted of loosening the adhesions.

During the subsequent year he had the same symptoms, which were unrelieved. He returned sixteen years ago, when he was x-rayed again and the irregularity of the duodenum was more pronounced than the first time. He was explored again and even more adhesions found at the second operation than at the first. There was still no evidence of ulcer found. The adhesions this time, according to the note from the hospital, were said to involve the colon. A gastro-enterostomy was done with complete relief of symptoms for over fifteen years.

About six months before admission he began to be bothered by gas, marked anorexia and almost constant midepigastric pain, non-radiating, unrelieved, and not related to anything that he ate or did. He also noticed marked constipation and severe attacks of retching, during which he did not vomit food but regurgitated small amounts of liquids. His family noticed increased pallor. During the subsequent three months the symptoms grew more and more severe. He was particularly bothered by constant retching and anorexia. Three months before admission he went to the Beth Israel Hospital, where he remained for a week and a diagnosis of carcinoma of the stomach was made. Inasmuch as it was felt that nothing could be done for him he was discharged.

He stayed at home, where all his symptoms gradually grew worse. He had increasing weakness and had to give up work. Retching was so severe that about six weeks before entrance he brought up approximately one ounce of bright red blood on one occasion. A month before entry he went to the Pondville Hospital, where he was studied and esophagoscopy was done, I am told. He had another hematemesis of about two ounces at the Pondville, and tarry stools. He was told that he did not have carcinoma of the stomach but duodenal ulcer. He was discharged on a diet.

He followed the diet, but gradually grew worse. Four days before admission he felt very weak. The night before admission he had a constant desire to defecate and while straining at stool had a tarry movement, felt weak and dizzy, and fainted. He remained in bed the next day. His doctor was called and advised admission to the hospital. The patient had lost about thirty pounds in all during the six months of illness.

His marital history is not contributory, but it is interesting that his wife and two of seven children are supposed to have had peptic ulcers.

His physical examination, except for the fact that he was very ill, pale, dehydrated and emaciated, was negative. The urine was negative. The blood pressure was 100/70. The pulse was 110, the respiration 20. The white cell count was 11,400, the hemoglobin 30 per cent Tallqvist, the red cell count 1,300,000.

He was admitted to the Medical service. Although his pulse and blood pressure were not exactly alarming, it was felt that because of the low red count he should be transfused. The surgeons were asked to transfuse him. There followed a long discussion as to whether he should be transfused. The medical men were for transfusion, the surgeons against it. Inasmuch as we on the Medical service were not doing our own transfusions at the time the patient was not transfused. The following morning however, after his blood pressure had sunk to about 80 and his pulse had risen somewhat, he was given a transfusion of about 500 cubic centimeters with marked improvement in the blood but no improvement in the general clinical condition. He remained in the ward ten days and was again transfused. At no time was he in sufficiently good condition to carry out diagnostic procedure of any sort. He continually had marked retching during which he would bring up small amounts of dark sanguineous material strongly positive for blood. He had very frequent attacks of uncontrollable hiccups. At times he had marked diarrhea during which he passed ten to twenty very small bowel movements during the day. All of these were strongly positive for occult blood but were never tarry. About the sixth or seventh day he developed a very marked suppurative parotitis on one side and the following day bilateral suppu-

*Assistant resident on the East Medical service.

rative parotitis; at the same time the temperature started to rise. The blood pressure remained between 80 and 100 systolic and the temperature ranged between 100° and 101°. The pulse was 100. On the tenth day he developed signs of a pneumonic process in both lungs, the temperature and pulse rose very markedly and he died.

Clinical Discussion

Dr. Tracy B. Mallory: This man at one time or another was in almost every Boston hospital. Through the kindness of Dr. Altman of the Beth Israel we have the plates taken when he was there. Perhaps he will show them to us.

Dr. William S. Altman: These are some of the films taken at the first examination, when it was noticed while fluoroscoping that he had an irregularity with definite cascading of the meal at what we at that time called the lower end of the esophagus; also another irregularity at the duodenum, thought to be due to perforating ulcer or to traction diverticulum secondary to old ulcer. We advised esophagoscopy, which was carried out and no lesion was seen. We were however quite positive that pathology was present, so we took him back to the X-ray Department and studied him very carefully. We noticed this time that the irregularity was definitely seen, but involved the cardia just below the cardio-esophageal junction. The irregularity was seen here, accompanied by a "splitting" of the meal which in our minds is sufficient to warrant a diagnosis of tumor mass at the cardia. That was our final diagnosis. In addition, we mentioned that he also had an irregularity of the duodenum, probably a duodenal ulcer.

Dr. George W. Holmes: As Dr. Kranes stated, the patient was not examined at this hospital. These films were taken at Pondville. The diagnosis that had been made at the Beth Israel was known and the radiologists at Pondville were asked to locate the carcinoma of the stomach which had been described. Two roentgenologists saw him with that definitely in mind and made two examinations, but were never able to demonstrate the lesion. Of course a lesion in that part of the stomach is extremely difficult to diagnose by x-ray; I think Dr. Robbins and his associates are to be complimented on being able to find it. In addition there is a variation from day to day in these cases due to the presence or absence of ulceration and spasm. I saw a patient this past week at the Phillips House who had been examined ten days previously for possible carcinoma of the colon and no lesion found. When I saw the patient the lesion was perfectly obvious. There was marked stricture with complete obstruction. The ulceration and spasm made it easier to demonstrate.

In these films the distance from the fundus of the stomach to the diaphragm is perhaps greater than it should be, and there is a suggestion of a tumor mass projecting into the stomach. Having seen the specimen and these films I have no doubt that this soft tissue shadow represents the lesion, but I should not expect any roentgenologist to make a diagnosis of carcinoma on such a finding. As has already been pointed out, the diagnosis in these cases is usually made with the fluoroscope rather than from the films. The cascade effect of the meal over the tumor is the best diagnostic point we have. Most tumors close to the cardia also cause obstruction at some time and we may get delay in the lower end of the esophagus, as you saw. In these films there is no evidence of delay.

This case also illustrates the fallacy of relying entirely on the x-ray examination. One should give greater consideration to symptoms.

Dr. Gerald Blake: This patient entered the wards after pretty thorough study at two other hospitals, with two different diagnoses and with a history of blood loss from the gastro-intestinal tract at varying times for seventeen years and an acute blood loss shortly before entrance. The diagnosis was not clear, but the case brings up very acutely the question whether to transfuse and when to transfuse. Where the diagnosis is definitely one of ulcer I do not think transfusion is so clearly indicated. Here, where the element of great blood loss comes in and there is a question of carcinoma of the stomach in a patient with a blood count and a blood pressure as low as this, it seemed to me that he deserved transfusion. We felt that whether or not there would be any effect on the bleeding aside from the improvement in his blood from the new blood put in, it was worth trying. I should like Dr. Bock's opinion as to the possible effects of transfusion on oozing areas which have caused a chronic blood loss. The patient did not respond satisfactorily, the bleeding continued, and a diagnosis of probable carcinoma of the stomach was made.

Dr. Arlie V. Bock: The question which Dr. Blake raises is a perennial one about this hospital. We have two schools of thought on the question divided into the right and the left group. I have always been on the side of those who feel that a case like this should have transfusion. The surgical feeling about it is that if we transfuse a bleeding patient all we do is to pour in blood that is immediately poured out through the oozing areas. There is however something to be said on the change of the coagulation time of the blood by adding five, six or seven hundred cubic centimeters of new blood to act as an increased means of stasis as opposed to the coagulation time of blood with a red count of 1,300,000. I think we have in our experience on the Medical side seen good results from transfusion in these cases and I have yet

to hear any reasonable arguments against doing it.

DR. MALLORY: One other diagnosis should, it seems to me, have been seriously considered in this case. Our only evidence of carcinoma at the cardia or the lower end of the esophagus was roentgenological. What they had at the Beth Israel was perfectly definite; no doubt there was a tumor there, but not necessarily carcinoma. The cardia is a favorite spot for benign tumor, and a history of bleeding for seventeen years is a very good history of benign tumor of the stomach. Even though you thought the chances were ten to one that it was carcinoma, I think a man with that story should have been explored on the chance that it was a benign tumor that was operable and curable. Would you not agree to that, Dr. Bock?

DR. BOCK: On account of the duration, yes.

CLINICAL DIAGNOSES (FROM HOSPITAL RECORD)

Duodenal ulcer.
Carcinoma of the stomach?
Generalized arteriosclerosis.
Acute bilateral parotitis.
Bronchopneumonia.

ANATOMIC DIAGNOSES

Adeno-acanthoma of the esophagus with metastases to the mediastinal glands.
Pulmonary thrombosis, left.
Pulmonary infarcts.
Bronchopneumonia.
Pulmonary edema.
Chronic fibrous pleuritis, right.
Arteriosclerosis, slight, aortic, coronary and renal.
Gastrojejunostomy.
Bilateral parotitis.

PATHOLOGIC DISCUSSION

DR. MALLORY: The tumor is exactly at the cardia. A small amount of it perhaps passes above the junction of the esophagus and the stomach, but the major part of the tumor is actually within the stomach. The border line between the stomach and the esophagus is a very irregular one and it is difficult to localize it exactly. The tumor was low enough so that I think it is not at all surprising that direct esophagoscopic examination was negative. One can do a biopsy without very much difficulty through the esophagoscope on a tumor that is in the esophagus, but when it is as low as this one might not be able to get down to it.

The histology is also somewhat interesting. We finally made a diagnosis of adeno-acanthoma. That is an adenocarcinoma showing areas of differentiation into squamous epithelium, a type of tumor that is very common in the uterus but less often seen anywhere else. Recently they have been shown to be not unusual in the gall bladder. We had one case in which we found an adeno-acanthoma in the sigmoid, but that patient had previously had a hysterectomy, so that it might well have been a metastasis.

DR. BOCK: Did he have an ulcer?

DR. MALLORY: There was a small ulcer in the duodenum.

DR. BOCK: How long do you think the tumor in the cardia had been present?

DR. MALLORY: I do not believe one can tell. It must have been present six months or more, very possibly two years, but I am guessing at those limits.

ERRATUM

IN Case 19032, published January 19, 1933, the hemoglobin readings on page 156 should be 22 per cent, 32 per cent, 24 per cent, 26 per cent and 27 per cent.

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THE TREATMENT OF BURNS

IT is not frequently that a medical journal has the opportunity of publishing a first paper by a recent graduate that records a fundamental and important advance in knowledge and, in addition, is extremely well written*. The authors of the manuscripts that flow into journals and have to be returned on account of poor writing, unimportant subject matter, non-critical deductions or many other reasons, could well afford to study this article and use it as a model. Unfortunately it would be too much to expect that every article should contain such important subject matter. The fact that this one does can well cause many surgeons, as it has the writer of this editorial, to kick himself mentally for not having thought of the important rôle played by infection with virulent streptococci in the course and outcome of that Cinderella of diseases, burns. It is interesting how frequently others have been in a position to make discoveries before or at the time that an advance in medicine has been made. Here is an instance where a few simple cultures, a method of study universally available for sixty years, plus a little thought and some very earnest detailed work, have changed the entire point of view of treatment and, as a result, the outlook for the great majority of the many victims of these frightful accidents. The editorial writer well remembers that when an interne he treated a series of cases with the then new ambrine treatment, and his chief, now retired, felt that the retention of pus under the paraffin for twenty-four hours at a time was harmful. It was also noticed that islands of skin that appeared to be viable on the first and second days would mysteriously melt away before a week had gone by. Of course, all granulating wounds were known to be infected, but nobody thought seriously enough about the infection to investigate it. These remarks are meant in no way to detract from the importance of the other two great advances that have taken place in this field in the last decade. First, the tannic acid dressing with its protective action was a great step in the way of local treatment. Secondly, the study of the blood chemistry, by Underhill, led to important advances in treating the shock of the first few days. This shock, often being present on the admission of the patient, must still be attended to. The replacement of tannic acid by aqueous gentian violet results in a softer, more stable eschar and, in addition, has very potent antiseptic properties without at the same time hindering the growth of cells. It goes without saying that this combination of properties is almost unique. In severe cases it is not and can never be a lazy man's method of treatment. Daily strict attention must be paid to the details of local and general treatment.

In closing we wish to congratulate the author for having made such fruitful use of his opportunities as a student, interne and surgical resident.

*Aldrich, Robert Henry: The rôle of infection in burns. New Eng. J. Med. 208:299 (Feb. 9) 1933.

"NO LEGISLATION NECESSARY"

IN the "Bulletin of Committee Work and Business of the Legislature" for March 6, 1933, opposite House Bill 106 and indicating its disposition by the Legislature are the words "No legislation necessary." Taken in connection with the situation as it exists, this does not make sense. As the Secretary of the Board of Registration in Medicine pointed out in discussing the bill, a change in the law is needed so that the graduates of the better schools can once more be registered in Massachusetts; and also, judging from the statements in the catalogues of medical schools, in the light of a recent opinion of the Attorney General, that practically the only persons eligible for examination in Massa-

chusetts are those who are refused examination in every other state.

Under the opinion of the Attorney General, the Board seems to be requiring specific information as to dates from each candidate concerning the course given by the school from which he received his degree, so that the Board may decide for itself whether the time qualification is fulfilled. Apparently it is not fulfilled by most schools. While the Board has made no public statement concerning all or any candidates rejected, certainly some graduates of Boston University, of Harvard and of Tufts have been told they are ineligible.

If no legislation is necessary, it means that the Legislature desires to exclude from examination and therefore from practice in Massachusetts the graduates of Yale, Columbia, Cornell, Pennsylvania, Jefferson, Johns Hopkins, Vanderbilt, Western Reserve, Chicago, Washington, Michigan, Stanford, California and other medical schools outside of Massachusetts of high reputation, as well as the graduates of Boston University, of Harvard and of Tufts. It is this that does not make sense. Perhaps the Legislature has made this disposition of House Bill 106, without realizing just what are the issues involved, but certainly the responsibility for the present situation rests squarely on the Legislature.

AN IMPORTANT BILL (HOUSE 343)

Representative Estabrook of Fitchburg appeared before a committee of the Legislature in advocacy of a bill to create a State Board which would have authority to investigate institutions which confer degrees and to establish standards of instruction by such institutions and if advisable recommend revocation of charters which have been conferred by this Commonwealth. Similar arguments were presented at a previous hearing by the Commissioner of Education, Mr. Payson Smith; the Commissioner of Public Health, Dr. George H. Bigelow; and the Secretary of the Board of Registration in Medicine, Dr. Stephen Rushmore.

At this previous legislative hearing the Middlesex College of Medicine and Surgery had asked for authority to confer a degree in Dentistry. This opened the subject of granting degrees by educational institutions and inspired Representative Estabrook to appeal to the Legislature for the appointment of a board with powers respecting this matter.

There is undoubtedly abundant reason for the creation of such a board, for charters have been given to many institutions, by the several states, which have not been free from criticism. With respect to the Board of Registration in Medicine, evidence has been submitted indicating that until it has authority to examine medical schools it cannot know whether applicants for registration have had that training which is generally required by the other registration boards of this country.

The Massachusetts Medical Society

COMMITTEE ON POSTGRADUATE MEDICAL INSTRUCTION

A meeting of this committee was held at the Society headquarters, 8 The Fenway, on March 6. There were present Dr. James H. Means, Dr. Charles W. Blackett, Jr., Dr. Joseph W. O'Connor, Dr. Dwight O'Hara, Dr. Reginald Fitz, Dr. Alexander S. Begg, Dr. Frank R. Ober, Dr. A. Warren Stearns, Dr. Walter P. Bowers and Dr. George H. Bigelow.

Dr. Frank R. Ober was elected chairman.

The executive committee, consisting of Dr. Greenough, Dr. Fitz and Dr. O'Connor, was duly elected.

Dr. Leroy E. Parkins has been chosen to serve the committee and the executive committee as secretary.

THIS WEEK'S ISSUE

Contains articles by the following named authors:

Davis, David. B.S., M.D. Cornell University Medical College 1925. Research Fellow, Thorndike Memorial Laboratory, Boston City Hospital. Address: 485 Commonwealth Avenue, Boston. Associated with him is

Weiss, Soma. A.B., M.D. Cornell University Medical College 1923. Associate Professor of Medicine, Harvard Medical School. Director of the Second and Fourth Medical Services (Harvard). Associate Physician, Thorndike Memorial Laboratory, Boston City Hospital. Address: Boston City Hospital, Boston. Their subject is "The Relation of Subacute and Acute Bacterial Endocarditis to Rheumatic Endocarditis: A Study of 66 Cases With Necropsies." Page 619.

Howe, Henry F. B.A., M.D. Harvard University Medical School 1930. Surgical House Officer and Surgical (Harvard Teaching) Service, Boston City Hospital. His subject is "Appendicitis." Page 624. Address: 231 Beacon Street, Boston.

Findlay, Francis M. A.B., M.D. Harvard University Medical School 1922. F.A.C.S. Assistant Surgeon to Outpatients, Massachusetts General Hospital. Assistant Surgeon, Cambridge Hospital. His subject is "Atropine as a Diagnostic Aid in Appendicitis." Page 630. Address: 475 Commonwealth Avenue, Boston.

WOODBRIDGE, PHILIP D. A.B., M.D. Harvard University Medical School 1921. Anesthetist, Lahey Clinic. His subject is "Better Gas Anesthesia. (The Carbon Dioxide Absorption Method.)" Page 632. Address: 605 Commonwealth Avenue, Boston.

WOODWARD, SAMUEL B. A.B., M.D. Harvard University Medical School 1878. President, Massachusetts Medical Society 1916-1919. His subject is "Smallpox and Vaccination." Page 641. Address: 58 Pearl Street, Worcester, Mass.

TAYLOR, ERWIN H. M.D. University of Michigan 1900. Consultant, House of Mercy and Hillcrest Hospitals, Pittsfield, Mass. His subjects are "Lymphosarcoma of the Jejunum" and "Ruptured Aneurysm of the Hepatic Artery." Pages 642 and 644. Address: Pittsfield, Massachusetts.

GUTHRIE, RILEY H. M.D. University of Tennessee School of Medicine 1921. Assistant Superintendent, Monson State Hospital, Palmer, Mass. His subject is "Some Recent Researches in Epilepsy at the Monson State Hospital." Page 646. Address: Palmer, Massachusetts.

BOSTON MEDICAL LIBRARY

THIS column in the *Journal* will, from time to time, give a list of the recent accessions to the Library and, without any attempt at trespassing upon the field of the reviewer, will offer in the case of the more significant books, a thumbnail sketch of the content of the volume and the author's approach to his subject, not with any idea of presenting an abstract of the subject but merely outlining the character of the work presented, whether clinical or philosophical, monographic or sketchy, in order that some suggestion of its importance, over and above what the title might indicate, may be conveyed.

In the following list of new books catalogued to January 5, 1933, there are quite a number of titles on subjects that are largely cultural and historical.

The subject of Medical Education is represented by three very important reports which should engage the attention of all men interested in the future of medicine, and just now very much in the "spotlight."

Surgery comes in for only eight to ten titles, some of which are concerned with technique and some with the surgical pathology of disease.

In Medicine the subjects include cancer, hookworm disease, Bright's disease, diabetes, preventive medicine, rheumatism, leprosy, dietetics and a number of somewhat more specialized conditions, and in a lighter vein there has been added a volume of essays: "Taking the Doctor's Pulse" and a story with the title of "Amok."

Cultural

Mystery and Art of the Apothecary. C. J. S. Thompson.
History of Scottish Medicine. J. D. Comrie.
History of the Massachusetts General Hospital. G. W. Myers.
One Hour of Medical History. Ed. 2. Benjamin Spector.
Hermes Hermetica. Hermes Trismegistus.
Medical Art Calendar. J. P. Kruseman.
Paediatric Biographies. J. Rühräh.
Surgery at the New York Hospital, One Hundred Years Ago. Pool & McGowan.

Educational

Reprints & Problems of Medical Education. Rockefeller Foundation.
Commission on Medical Education, Final Report.
Committee on Costs of Medical Care. Final Report.
Medicine and The State.
Fads & Quackery in Healing. Fishbein.

Surgery

Practical Surgery of the Joseph Price Hospital. J. W. Kennedy.
Diabetic Surgery. McKittrick & Root.
Orthopaedic Surgery. (See review in Journal of Bone & Joint Disease, October, 1932). W. Mercer.
The Operating Room. A. A. Smith.
Surgery with Special reference to Podiatry. E. Adams.
Laboratory of Surgical Practice. Dodds & Whitby.
Let's Operate. McKay & Beasley.
Recent Advances in Anesthesia and Analgesia. C. L. Hewer.
Gynäekologische Operationslehre. J. Halban.
Taking the Doctor's Pulse & another essay. J. F. Montague.
Amok. A story. S. Zweig.

Internal Medicine

Hot Mineral Baths Commission—Conference on Rheumatic Disease. Bath City Council.
Cancer and its Allied Diseases. W. F. Koch.
The Mass Diagnosis of Hook Worm Disease. C. Lane.
Diabetes in Childhood and Adolescence. P. White.
Clinical Dietetics. H. Gauss.
Synopsis of the Practice of Preventive Medicine, etc., and Clinical Instruction at the Harvard Medical School. S. Warren.
Psychological Effects of Menstruation. M. Chadwick.
The Renal Lesions of Bright's Disease. Addis & Oliver.
Disease and the Man. G. Draper.
Manual for the Jewish Diabetic. W. S. Collins.
The Rheumatic Infection in Childhood. (See Review in Journal of Bone & Joint Disease, July, 1932.) L. Findlay.
The Endocrine Disorders—Differential Diagnosis in: A. W. Rowe.
Studies on Hookworm. Ascaris and Trichuris in Panama. W. W. Cort et al.
Leprosy—Survey in 40 countries. H. C. de Souza-Araujo.

MASSACHUSETTS LEGISLATIVE NOTES

House bill No. 1239 By Mr. Hays of Boston, petition of Morris A. Cohen for incorporation of Alice Stone Blackwell and others as the Reconstruction Clinic with authority to maintain a medical clinic for care of sick and disabled persons. Public Health.

House bill No. 926, Petition of Joseph A. Conway for certain amendments of the law relative to registration and practice of chiropractors, will be heard March 28, before the Committee on Public Health, Room 450, at 10:30 A.M.

MISCELLANY

A RADIO MESSAGE PREPARED AND SPONSORED BY THE COMMITTEE ON PUBLIC EDUCATION OF THE MASSACHUSETTS MEDICAL SOCIETY FOR THE DEPARTMENT OF PUBLIC HEALTH

THE EFFECTS OF FATIGUE*

BY JOHN H. TALBOTT, M.D.

There are two significant types of fatigue that may be subjectively experienced by the human individual. These are, in the order of discussion, physical and functional fatigue. The first type is usually associated with physical exertion and the sensation is referred to the voluntary muscles. If the work has been strenuous, the physiological changes that occur in the body may be quantitatively analyzed. The respiratory rate and heart rate will be increased with an added uptake of oxygen. The concentration of red and white cells in the blood likewise will be increased. There is another constituent of the blood that is altered in exercise that has recently been the subject of considerable study; this is an organic acid called lactic acid. It has been found that the concentration of this substance increases many fold in the muscles in the active state, and with the accumulation of lactic acid in the muscles there may be a spilling over into the blood stream. When lactic acid enters the blood stream it competes with a weaker acid, carbonic acid, and lowers the carbon dioxide content of the blood. Thus it can be said that the important changes in the blood following muscular work are an increase of the red and white cells and lactic acid, and a lowering of the carbon dioxide combining power. But it is not possible at present to go farther and say that one or all of these changes are responsible for the subjective sensation of physical fatigue.

The other type of fatigue that I wish to discuss is mental or functional. The division between physical and functional fatigue is not always exact, nor is it necessary in this instance that it should be. After a day's work of either physical or mental labor we may experience similar subjective sensations. While the blood changes may be found after the physical labor, no significant changes are observed after mental labor. This evidence points against the known chemical changes as being primarily responsible for the subjective sensation of fatigue. It should be noted that while there may be important chemical changes in the cells of the nervous system in either type of fatigue, proof for this is not at present conclusive.

*Station WBZ, February 28, 1933, 4:15 P.M.

The assignment of seven or eight hours of work daily for most people in the social order today presupposes the ability of the average person to carry on for this period of time before the onset of fatigue. Some people however are not able to follow such a schedule, either the whole or in part. To these individuals who are chronically tired the term neurasthenics has been given. But it should be said at this time that while fatigue in these persons is generally an emotional reaction, fatigue may be a symptom of a disease requiring medical advice and care. Among the diseases in which fatigue is an early and significant symptom are Addison's disease, myasthenia gravis and effort syndrome. Of more widespread experience is the feeling of fatigue that may be the onset of or accompany many acute and chronic diseases. The awareness of increased fatigability in an individual with a stable nervous system is a danger signal that should be promptly heeded.

After recognizing the sick person with organic disease and the neurasthenic who is chronically exhausted, let us consider the individual with a capacity for work greater than the average. Napoleon, Poincaré, Edison and Theodore Roosevelt were men with such a reputation. Physiological data on such individuals are not at hand and casual observations are difficult to evaluate. In the absence of quantitative data on these persons, let us examine the facts already recorded on certain athletes and their capacity for work. As an example, members of the world championship Yale crew for 1924 were able to row for a six-minute period using oxygen at a rate about 20 times the resting value. A Marathon race is another severe test of physical effort. In the twenty-six-mile Boston Marathon, a runner may consume more fuel in three hours than many laborers consume in a full day's work.

What accounts for the great difference in performance in the neurasthenic person who is chronically exhausted and the Marathon runner who may run eight to ten miles four times a week for months in preparation for a race? Constitutional endowment at birth may favor the man engaged in muscular work. Yet many athletes may be physically normal without being physically perfect. The concentration of hormones in the body at any given time may be important. It has been known since the investigations of Dr. Cannon that in acute emergencies the hormone adrenalin may be increased and aid the person in adequately meeting a given situation. This increase of adrenalin may be significant early, in a long contest, but its effect probably is unimportant after this time.

There is another hormone from the adrenal gland only recently isolated that may be equally important in the prevention of fatigue in various individuals. The name of this hormone is cortin. Professor Hartman at Buffalo has found that cortin is decidedly beneficial when given to neurasthenic patients. This observation suggests a pos-

sible deficit of the hormone in this group of patients.

A third and equally important factor responsible for the differences in individual achievement is the speed of chemical processes in the body. At present indirect proof only is available confirming this assumption. Continued study of the physiology of muscular exercise and of the pathological physiology in the thyroid disease may further substantiate this hypothesis.

But studies of the constitution, hormones and chemical processes do not fully explain the great variation in onset of fatigue in the neurasthenics and the athletes. Further investigation may associate it more clearly with one of the mentioned factors. In the absence of conclusive data let us examine certain accepted findings. In a period of prolonged patriotic stress as in 1917-1918, production of raw material reached a magnitude that was merely dreamed of ten years before. The increased production of the nation was a direct function of the increased production of the individual. In the language of the runner, the nation had found its "second wind".

Professor William James in his interesting paper in the Philosophical Reviews of 1906 entitled "The Energies of Men" says, "If an unusual necessity forces us to press onward, a surprising thing occurs. The fatigue gets worse up to a certain point, when gradually or suddenly it passes away, and we are fresher than before. We have evidently tapped a level of new energy, masked until then by the fatigue obstacle usually obeyed. There may be layer after layer of this experience. Mental activity shows the phenomenon as well as physical and in exceptional cases we may find, beyond the very extremity of fatigue-distress, amounts of ease and power that we never dreamed ourselves to own, sources of strength habitually not taxed at all because habitually we never push through the obstruction, never pass those early critical points." This is not the energy of youth, but the energy of mature age. The disturbing sociological phenomena during the years since the war should not be responsible for the onset of fatigue in this generation and period of world history. The fact is, however, that the wear and tear of modern life, made up of tension in business, worry, domestic difficulties and similar factors, are important causative agents in excessive fatigue. An intelligent understanding of these sociological phenomena would go far toward fortifying the individual against them.

In summary then it may be said that fatigue may be a useful physiological and psychological state but we need not become sensitized to it.

MORTALITY RATES

Telegraphic returns from 85 cities with a total population of thirty-seven million for the week ending March 4, indicate a mortality rate of 11.5 as against a rate of 13.4 for the corresponding week of last year. The highest rate (17.9) appears for Memphis, Tenn., and the lowest (5.4) for Yonkers, N. Y. The highest infant mortality rate (13.7) appears for Lowell, Mass., and Schenectady, N. Y., and the lowest for Cambridge, Mass., Lynn, Mass., Peoria, Ill., and Trenton, N. J., which reported no infant mortality.

The annual rate for 85 cities is 12.5 for the nine weeks of 1933, as against a rate of 12.3 for the corresponding period of the previous year.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR FEBRUARY, 1933

The low prevalence of communicable disease noted last month has continued.

The number of cases of diphtheria has dropped to a remarkably low figure, being less than half of the number of cases reported the same month of last year.

Only four cases of typhoid fever were reported, which is a new record for this disease for any month of the year.

Anterior poliomyelitis and epidemic cerebrospinal meningitis both likewise established new low records, each having only one case reported this month.

This is the tenth consecutive month in which the number of cases of pulmonary tuberculosis reported has been the lowest for that particular month.

The prevalence of measles has been unusually low.

Lobar pneumonia, which showed a considerable increase last month, has again dropped to quite low levels.

Scarlet fever continues its decline from the peak which it reached last season.

Whooping cough, German measles, mumps, influenza and non-pulmonary tuberculosis were reported about the number of times expected.

RARE DISEASES

Anterior poliomyelitis was reported from Watertown, 1.

Anthrax was reported from Haverhill, 1; Lynn, 1; Peabody, 1; Total, 3.

Encephalitis lethargica was reported from Agawam, 1; Dartmouth, 1; Gardner, 1; Northampton, 1; Total, 4.

Epidemic cerebrospinal meningitis was reported from Gardner, 1.

Malaria was reported from Brockton, 1; Taunton, 1 (Therapeutic); Total, 2.

Pellagra was reported from Worcester, 1.

Septic sore throat was reported from Boston, 9; Braintree, 1; Chelsea, 1; Dover, 1; Everett, 1; Hingham, 2; Ipswich, 1; Malden, 1; Medford, 1; Newburyport, 1; Newton, 2; New Bedford, 1; Northampton, 2; Quincy, 5; Revere, 4; Somerville, 1; Topsfield, 5; Weymouth, 1; Total, 40.

Trachoma was reported from Boston, 1; Brookline, 1; Lynn, 1; Marblehead, 1; Total, 4.

Trichinosis was reported from Cambridge, 1; Chelmsford, 1; Lowell, 11; Lynn, 1; Somerville, 2; Tewksbury, 4; Wakefield, 1; Total, 21.

MONTHLY REPORT FOR FEBRUARY, 1933

Disease	Feb. 1933	Feb. 1932	Average*
Ant. poliomyelitis	1	4	5
Chicken pox	1106	914	1002
Diphtheria	96	223	323
Dog Bite	319	291	267
Epidemic Cerebrospinal Meningitis	1	6	10
German Measles	51	62	140
Gonorrhea	427	525	421
Lobar Pneumonia	466	489	680
Measles	982	1565	2892
Mumps	718	1168	851
Scarlet fever	1501	2065	1442
Syphilis	340	308	247
Tuberculosis Pulmonary	220	276	371
Tuberculosis other forms	35	31	45
Typhoid Fever	4	13	13
Whooping Cough	732	854	868

*Average number of cases for February during the preceding five years.

EXCERPTS FROM THE *BULLETIN OF MIDDLESEX COLLEGE OF MEDICINE AND SURGERY* —1932-1933

"This College . . . is unique . . . Massachusetts has an unusual number of excellent institutions of learning. All of them do what they can for poor but worthy students. Few of them can do much. None of them specialize in students of that sort. We do just that."

. . . . . .

"The college welcomes the poor, intelligent, studious boy and the youth of moderate means. It has no endowment, it can do little financially for the former, perhaps it is better that it cannot help in this way."

. . . . . .

"It recognizes the 'Mark Hopkins' element and its importance. It seeks its endowment in scholarships, that there may be students to put on the log with Mark Hopkins."

. . . . . .

"The course of instruction differs from those of other colleges in the added provision that students are given study assignments week by week in the required textbooks. The assigned studies correspond as nearly as possible with the subjects being taught at the time in the class rooms and laboratories. Written tests on this work are given about once a month."

. . . . . .

"Students may be admitted to advanced standing upon credits from other medical schools when vacancies occur, but it is understood that because of the full enrollment there are comparatively few of such students accepted."

. . . . . .

"Heretofore the facilities of the college have limited the enrollment to between 55 and 75 students per class or not to exceed 300. Every year a certain proportion have failed of acceptance because of this restriction."

The enrollment in the Fourth Year Class was, nevertheless, only 52, leaving 23 places for students to be admitted to advanced standing if the *enrollment were not full.* Similarly there were 21 places, in the third year, and 20 places in the second year to which students could not be admitted to advanced standing as the *enrollment was so full.*

"In the interest of economy this catalogue does not publish the usual uninteresting, encyclopedic, and redundant description of courses of study, prefaced with long lists of professors. These courses are practically the same in all medical schools, including Middlesex. Anyone desiring such a description should consult any one of the usual conventional catalogues."

ALFRED ELA

A LAYMAN VERSED IN MEDICAL LORE

Born in Washington, D. C., of old New England stock seventy-six years ago, Alfred Ela received his early education at the Cambridge High School and Phillips Exeter Academy followed by a few years of study at Harvard College and in Germany. At the age of about 40 years he began to take an interest in literary investigation, in folk lore, and archaeology and in the history of medicine and through the years ever since while carrying on his own business, he has continued this interest, and has taken an active part himself as evidenced by various letters and articles that have come from his pen. A few examples among these contributions are the following:

Napoleon's wounds. Correspondence. Boston M. & S. J. 174: 813, 889 (June 1) 1916.

Across a gap of 2,000 years. Correspondence. J. Am. Vet. M. A. 48 (New series I): 650, 1916.

The coventry case. Correspondence. Boston M. & S. J. 179: 747 (Dec. 12) 1918.

Ela, Alfred: Some forgotten nativities. Am. J. Obst. & Gynec. 6: 501, 1923.

Beginning thirteen years ago Mr. Ela's eyesight troubled him and gradually he grew blind so that for more than ten years now he has been unable to read. Before this time he had always led an active outdoor life. Shortly after his blindness became extreme, important heart trouble developed and this has also been a serious handicap. Uncomplainingly and with rare courage and cheerfulness Mr. Ela has not only faced these two infirmities but has continued actively at work, going out and spending many hours in his office every day, despite his advancing years. He is a faithful attendant at the regular meetings of the Boston Medical History Society and generously contributes information to anyone who needs it from his own vast store. For years not an occasion has gone by when, on paying me a medical

visit, Mr. Ela has not left with me some notes, references or advice of value about my own work or my special interests.

I would pay tribute herewith to Alfred Ela, as a patient, a teacher, and a friend, for his courage and his character, for his knowledge and interest in medical science and history, and for the contributions that he, as a layman, has made. He is still an active coworker with us and I wish him to realize how much we value the example he has set us.

PAUL D. WHITE.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1932 AND SEVEN YEAR AVERAGE

MONTH ENDING MARCH 4, 1933

Diseases	1933 Week ending Feb. 11	Week ending Feb. 18	Week ending Feb. 25	Week ending Mar. 4	Average cases reported for week corresponding to Mar. 4 for past seven years	1932 Week ending Feb. 13	Week ending Feb. 20	Week ending Feb. 27	Week ending Mar. 5
Cerebrospinal Men.	2	—	1	1	2	—	2	3	—
Chicken Pox	100	96	94	105	94	145	137	92	212
Conjunctivitis Inf.	—	—	—	—	—	1	—	—	—
Diphtheria	3	3	6	1	22	6	9	8	7
Dysentery Bacillary	1	4	—	—	—	—	4	—	—
Encephalitis Epid.	1	—	—	—	—	2	1	—	—
German Measles	1	2	4	5	35	6	12	6	3
Influenza	87	38	24	24	21	9	21	24	20
Measles	148	159	189	178	410	207	278	301	283
Mumps	72	45	72	101	86	64	106	74	95
Paratyphoid Fever	—	—	—	—	—	—	1	2	—
Pneumonia (Broncho)	49	32	38	48	46	32	42	38	54
Pneumonia (Lobar)	63	38	43	37	44	44	51	51	84
Poliomyelitis	—	—	—	—	—	—	—	1	—
Scarlet Fever	98	97	137	109	96	71	112	129	143
Septic Sore Throat	3	—	—	—	4	—	10	17	3
Smallpox	2	1	—	2	—	12	2	3	2
Tetanus	1	—	—	—	—	—	—	—	—
Trachoma	—	1	—	—	—	—	—	1	1
Trichinosis	2	—	1	1	—	—	1	—	—
Tuberculosis (Pul.)	18	26	13	29	29	22	28	17	2
Tuberculosis (O. F.)	—	1	—	—	2	3	2	3	1
Typhoid Fever	2	—	—	—	1	1	1	1	—
Undulant Fever	2	1	—	1	—	—	2	—	—
Whooping Cough	78	82	36	118	76	134	129	90	15
Gonorrhea	37	21	66	51	31	28	77	35	4
Syphilis	69	40	54	54	45	42	63	30	5

Remarks: No cases of Asiatic cholera, glanders, plague, or yellow fever during the past seven years.

THE OVERSUPPLY OF PHYSICIANS*

The apprehension has been expressed that the reduction in the number of medical schools will lead to a shortage of physicians in this country. A study of the immediate past indicates rather the contrary and that there is larger production than necessary, and at the present time there is an oversupply of physicians in this country. The future supply is governed by a number of factors, some of which are largely problematical. The changes in primary, secondary, and college education may in the future allow medical students to graduate at an earlier age.

A special problem has been introduced in recent years by the large number of American students studying in the medical schools of Europe. At the present time the addition to the profession each

*Abstracted from the Final Report of the Commission on Medical Education, New York, Office of the Director of Study, 1932.

year from all sources is probably between 5,200 and 5,400 physicians which will probably be higher in the future, particularly because of American students studying abroad who will return to practice in this country.

It is estimated on the present ratio of increase that there will be 171,700 physicians in 1940, 186,600 in 1960, and 210,800 in 1980. At the present time the number of persons per physician in the United States is approximately 780. In 1940 it will be 760, in 1960 about 730, and in 1980 just under 700.

An adequate medical service for this country could probably be provided for by about 120,000 active physicians, if they were distributed in relation to the medical needs and their efforts were properly correlated. On such an assumption, there is an oversupply of at least 25,000 physicians in this country. If the present rate of supply is continued, the number of physicians in excess of indicated needs will increase.

The effects of an oversupply of physicians on the quality of medical service and indirect influence on the type of student seeking admission to medical schools is deserving of serious consideration. Those responsible for medical education and for the licensure of physicians should have this situation clearly in mind.

AN HONOR TO DR. HARVEY CUSHING

The *Boston Sunday Herald* of March 19, 1933, reports that Dr. Harvey Cushing was among five foreign savants named at The Sorbonne to receive the degree of doctor honoris causa from the Paris University which was approved March 18 in the ministry of national education.

VISION OF AUTOMOBILE DRIVERS

More thorough vision testing for applicants for automobile drivers' licenses, as a means of preventing accidents, was advocated by Maxwell Halsey, Traffic Engineer of the National Bureau of Casualty and Surety Underwriters, in an address yesterday afternoon before the session on "The Eye" at the Fourth Annual Greater New York Safety Conference in Hotel Pennsylvania. The session was sponsored by the National Society for the Prevention of Blindness.

"Present examinations," said Mr. Halsey, "covering color blindness, visual acuity and field vision, do not necessarily tell whether the operator will or will not have accidents. There is a considerable demand for improvements in eyesight examinations which will not only tell what picture the eye sees, but what effect this has in the brain of the operator.

"It is suggested that eyesight tests cover such items as the ability of the operator to recognize a hazard; the timing of the operator (his ability to judge speed and distance); the ability of the operator to see 'the whole picture' and not merely one part of it; and the reaction time of the operator to pictures which show a hazard.

"The Motor Vehicle Commissioners have done very well with present examinations, considering the facilities at hand. Millions have been spent on high speed roads, but only thousands have been spent on the field of safety. The greatest demand today is to get into actual practice what we already know.

"Only 16 states have a drivers' license law; and only 10 of these, including New York, New Jersey and Pennsylvania, have a good one. There can be no question but that drivers' license laws and drivers' examinations have reduced accidents. Those states using them have produced a 29 per cent reduction in accidents. States without such a law show an increase in the number of accidents."

Speaking on the eye examination of industrial employees, Dr. M. Davidson, Ophthalmologist of the New York State Department of Labor, recalled that the safety movement began with the discovery of color blindness as the major cause of railway and marine disasters.

CORRESPONDENCE

STATE MEDICINE FOR THE INDIGENT

Dear Editor,

In both the majority and the minority reports of the recently disbanded Committee on the Costs of Medical Care there is one point of agreement that has not received due emphasis: namely, that the government should provide medical care for the indigent. Now this class even at the *fastigium* of the glorious era of prosperity was admittedly by no means inconsiderable. Today, it is obviously inordinately large. Both the present economic conditions and the future prospects portend that it will inevitably remain big enough to demand our serious attention.

How is it to be met?

Surely not by continuing to educate and train young men and women to serve gratuitously in clinics and hospitals eventually augmenting a profession of which, according to the Report of the Committee on Medical Education, there is already in excess of the needs of our country no less than twenty-five thousand!

Nor by tacitly allowing further invasion of the legitimate field of doctors, particularly in preventive and industrial medicine, by members of the nursing profession with its surplus of more than thirty thousand!

Nor, it is to be hoped, by contract individualistic medicine, over which in a convention the secretaries of the state societies and the editors of regional medical journals wrangled for hours filling with their discussions forty pages of a Bulletin of the Americal Medical Association without getting anywhere!

Nor, God forbid, by small groups of doctors subject to the vicissitudes of petty politics and deprived of opportunities for self-realization or self-development!

How, then, can this harassing problem be solved but by a well-organized statewide medical service, manned and managed, trained and recruited, wholly by doctors, with the necessary assistance from ancillary personnel?

G. W. HAIGH, M.D.
242 Burncoat Street,
Worcester, Mass.

A HYPOTHETICAL EXPLANATION OF THE SLOWING OF THE FETAL HEART BEAT

March 8, 1933.

Editor,
New England Journal of Medicine,

During normal labor the fetal heart beats about 120 to 160 times a minute, the rate falling approximately 20 to 80 beats during uterine contractions. The reason for this normal retardation of the fetal heart rate, in labor, during uterine contractions has two commonly accepted explanations.

Schultz, in 1871, mentions as a cause for the fetal heart slowing with each contraction during labor, the compression of the fetal head.

The other common explanation has been the impairment of the placental circulation, causing circulatory imbalance in O-CO_2, and thus affecting the heart rate and rhythm, because of the increased CO_2 content of the blood.

That a rise in intracranial pressure is the cause of the difference in fetal heart rate during a contraction does not seem plausible. In the usual case no very definite difference will be noted in the fetal heart rate during any of the presentations and positions of the fetus in utero, providing all things are equal, as to no prolapsed cord, tetanic contractions, etc.

According to the physiologists, an excess of CO_2, when of moderate amount, quickens the heart. In very large amounts CO_2 poisons the bundle of His, produces heart block and a slow ventricular rate. It is hardly tenable that the momentary (from 30 to 60 seconds) compression of the placenta would cause anything more than a stimulating dosage of CO_2 to accumulate in the fetus, and this should accelerate, instead of retard, the fetal heart rate. Under normal conditions the asphyxia phase, i.e., poisoning of the bundle of His in the fetus in utero by CO_2 seems remote.

It is a known physiological fact that the depressor nerve when stimulated by stretching of the heart musculature and aorta reflexly slows the heart by increasing vagus tone. The depressor nerve protects the heart against the evil effects of any sudden rise of blood pressure, should this occur under resting conditions.

It appears tenable that the slowing of the fetal heart during a contraction, in normal labor, may be attributed to a defense mechanism. By the compression of the placenta during a labor pain, blood normally contained in this organ is placed into the fetal circulation, through the umbilical cord, thus increasing the blood volume and blood pressure normally found in the fetus under resting conditions. Dilatation of the heart and aorta, consequent upon the increased volume of blood, stimulates the depressor nerve of the heart. The depressor nerve increases vagus tone, slowing the heart.

Yours truly,

M. A. CASTALLO, M.D.
255 Thayer Street,
Providence, R. I.

AN ANSWER TO COMMENTS ON A PREVIOUS LETTER

New England Journal of Medicine,
To the Editor:

In my letter, published recently in the *Journal*, I supposed I had made myself clear regarding my belief in the efficacy of vaccination against smallpox and my faith in the ability of the medical profession to carry their point of view without the aid of legislation, as they are doing in typhoid, diphtheria, etc., and that further discussion was unnecessary. Recent editorials and letters appearing in your publication and in my mail would indicate either misunderstanding or unfair interpretation of my point of view.

No longer does medicine alone hold the reins in the realm of health, from the angle of the rank and file of our people. We have as supposed competitors osteopathy, Christian Science and others. It is entirely within the range of possibility that, should Christian Science, during the next twenty-five years, gain in numerical strength and physical assets, as it has in the past twenty-five years, it might be successful in putting on our statute-books legislation preventing the practice of vaccination. Even at this day they seem to meet with some success in preventing further vaccination legislation.

Should that day ever arrive, the writer's point of view regarding personal rights and liberties would receive more sympathetic understanding among his colleagues in the medical field than it does at present. Let us hope that, before that time arrives, man in his infinite wisdom will have learned the secret of maintaining health without the agencies of Vaccinations, Tonsillectomies and Cascara, and more tolerance of the other fellow's honest point of view.

J. WALTER SCHIRMER, M.D.
March 15, 1933,
520 Beacon Street,
Boston, Mass.

RECENT DEATHS

JOHNSON — ORVILLE EDSON JOHNSON, M.D., of Winthrop, Mass., died at a Boston hospital March 16, 1933.

Dr. Johnson was born at Chicopee Falls, September 13, 1867, the son of the Rev. Charles T. Johnson and Elizabeth Edson Johnson. His premedical

education was acquired at the Wesleyan University and he graduated from the Harvard University Medical School in 1894. He joined the Massachusetts Medical Society in 1894 and had practiced medicine in Winthrop until his death.

He served on the staff of the Whidden Hospital of Everett and was town physician of Winthrop for several years and a former member of the Winthrop School Committee. He had many civic and social affiliations.

Dr. Johnson leaves a widow, Edith Allen Johnson; a son, Thurston Allen Johnson; a daughter, Miss Gladys Johnson; a brother, Wilbur C. Johnson, and a sister, Mrs. Frank Bakeman.

THOMPSON — Edward Charles Thompson, M.D., died at the home of his daughter in Williamstown, March 6, 1933, aged 78. He was born in Hull, England, and received his medical education in London, coming to America in 1892. He practiced in Roslindale for forty years and served as a Boston School physician for sixteen years. He was also physician to the Sons and Daughters of St. George and the German Workmen's Sick and Death Benefit Society. He retired from practice last October.

Dr. Thompson leaves a widow, Dr. Clara L. Thompson; a daughter, Mrs. Leo N. Beverly, and three grandchildren.

COLBURN — Harry Hayford Colburn, M.D., of 103 Mount Vernon Street, Boston, died March 12, 1933, after a long illness.

He was born in Orono, Maine, in 1871, the son of James J. and Sarah Pendleton Colburn. His preliminary education was acquired at Westbrook Seminary and Bowdoin College and he graduated in medicine from the Bowdoin Medical School in 1895. He was especially interested in neurology and psychiatry and had served as assistant neurologist at the Boston Dispensary, and at the Suffolk County Jail for sixteen years.

Dr. Colburn was a Fellow of the Massachusetts Medical Society and the American Medical Association. He leaves a widow, Mrs. Mae Haley Colburn; two sons, Hardy Ross Colburn and Harry Hayford Colburn; four daughters, Mrs. A. E. K. Billings, Jr., Mrs. Marjorie L. Colburn, Miss Elinor M. Colburn and Miss Phyllis M. Colburn; also three sisters, Mrs. Arthur L. Fernald, Mrs. Eugene O. Loomis and Miss Caro F. Colburn.

DOWNING — Franklin C. Downing, M.D., of Stockbridge died March 18, 1933. He was born in Sodus, New York, in 1866, graduated in medicine from the Columbia University College of Physicians and Surgeons in 1896 and joined the Massachusetts Medical Society in 1897. He had practiced in Southbridge since 1916 and was formerly medical examiner. He was interested in civic affairs having served on the local board of health and as trustee of the Berkshire Farm School. He leaves a widow, a daughter and a son.

OBITUARY

RESOLUTIONS OF THE NEW ENGLAND ROENTGEN RAY SOCIETY ON THE DEATH OF DR. LAWRIE B. MORRISON

Dr. Lawrie B. Morrison died in Boston, January 16, 1933, of epidermoid cancer caused by repeated exposure to the roentgen rays. By Dr. Morrison's death the New England Roentgen Ray Society loses one of its charter members and a member devoted in attendance. Formerly President, he had continuously aided the Society and its members by his wise counsel and guidance.

Born in Vermont and educated at Peacham Academy and the University of Vermont Medical School, he came to Boston in 1914 to become an assistant to Dr. Walter Dodd. The death of Dr. Dodd from x-ray cancer in 1916 ended this happy association and Dr. Morrison carried on alone, continuing the principles and practices of his great preceptor. He slowly but surely built up that solid foundation on which his work now stands, a reputation for unusually accurate work, for a tireless energy, for remarkable attention to detail and above all for the enthusiasm with which he helped others, often at his own expense, modestly disclaiming all credit.

In his later years, with the judgment born of his tremendous experience and wide reading, he came to be more than a roentgenologist—he was known and acknowledged for his general diagnostic skill and most of the physicians who consulted him desired not only the report of his examination but also his opinion and advice as to the clinical problem presented by their patients. In short he was the finest type of general practitioner, specializing in diagnosis with the help of the roentgen rays and with unusual skill not only technical but also of that intangible variety which amounts almost to a sixth sense.

His death was the direct result of the meticulous and scrupulous care with which he made his examinations. Forewarned by the suffering and death of his preceptor he might justifiably have been satisfied with less exact results, but in his endeavors to give his patients the utmost in accuracy he deliberately transgressed the limits of safety and eventually gave his own life, an offering on the altar of science, and died a martyr to his own zeal.

Therefore be it resolved that the New England Roentgen Ray Society express publicly their warm esteem for Lawrie B. Morrison, their appreciation of his help and support in the organization and direction of this Society and in the promotion of roentgenology everywhere.

Merrill C. Sosman, M.D.

NOTICES

CLINICS IN MEDICINE AT PETER BENT BRIGHAM HOSPITAL

On Thursdays at 3:30 P.M. in the Amphitheatre of the Peter Bent Brigham Hospital Dr. Henry A. Christian, Hersey Professor of the Theory and Practice of Physic, Harvard, and Physician-in-Chief, Peter Bent Brigham Hospital, will give a series of clinics which members of the medical profession are cordially invited to attend. The next clinic will be given on March 30. There will be no clinic on Thursday, April 6.

Physicians and medical students are also invited to attend ward rounds of the Medical Staff of the Peter Bent Brigham Hospital held from 10-12 o'clock on Saturdays, as well as a clinical-pathological demonstration immediately following these rounds.

RADIO HEALTH MESSAGES

MARCH, 1933

Sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

Courtesy WBZ. Tuesdays, 4:15 P.M.

March

28 Hobbies and Health

STATE HOUSE BROADCAST

Sponsored by the Massachusetts Department of Public Health.

Courtesy WEEI. Fridays, 12:45 P.M.

Health Subjects of Public Interest discussed by the Division Directors of the Department.

RADIO HEALTH FORUM

Queries from the public are answered under the sponsorship of the Department of Public Health.

Courtesy WEEI. Fridays, 4:50 P.M.

Questions on Health and Prevention of Disease may be sent to Radio Health Forum, State Department of Public Health, State House, Boston.

HARVARD UNIVERSITY

FREE PUBLIC LECTURE

The Faculty of Medicine of Harvard University offers a free public lecture to be given at the Medical School, Longwood Avenue, Boston, on Sunday afternoon, March 26, by Dr. W. G. Smillie, subject, "A Discussion of the Common Cold." The lecture will begin at four o'clock and the doors will be closed at five minutes past the hour. No tickets are required.

MASSACHUSETTS GENERAL HOSPITAL

FRACTURE MEETING

There will be a conference under the auspices of the Fracture Service, Friday evening, March 24, at 8 o'clock, in the lower Out-Patient Amphitheatre.

SUBJECTS

Pathological Fractures. Dr. Harmer.

Fractures and Dislocations of the Cervical Spine. Dr. Roberts.

Report of Committee on Measurements of Joint Function and Rating of End-Results.

House officers, students and visitors welcome.

PHILIP D. WILSON, M.D., *Associate Chief.*

REPORTS AND NOTICES OF MEETINGS

THE HARVARD MEDICAL SOCIETY

The Harvard Medical Society held its stated meeting in the Amphitheatre of the Peter Bent Brigham Hospital February 28. Dr. Elliott C. Cutler presided. The meeting opened with a presentation of cases.

The first was a 58 year old female who entered the hospital with a complaint of swelling of the face and legs of two years' duration. These conditions were much improved with digitalis only to reappear six months ago. The patient has noticed that she prefers warm weather, that her skin is dry, and her hair is coarse and dry. Physical examination showed coarse, dry hair and dry skin. The face was puffy, and the subcutaneous tissues quite thick with pitting of the ankles. The heart was enlarged, and the blood pressure 230/100. There was a soft, freely movable mass in the mid-abdomen. X-ray films showed this to extend from the pelvic brim above the umbilicus. The basal metabolic rate was —34.

The second patient was a 50 year old female, who entered the hospital because of her debilitated condition. She suffered with a gluteal abscess eight months previously, which had ruptured the day before she appeared at the O. P. D. Débridement was performed under gas anesthesia, and because of weakness she was admitted to the house. Besides the debility, physical examination disclosed a freely movable, non-tender, elliptical mass in the left upper quadrant. Dr. Sosman, in commenting upon the x-ray studies with the barium meal in this patient, stated that they were quite typical of the condition of trichobezoar. They showed a non-opaque foreign body filling the stomach. This might also be a mass of shellac. At operation it proved to be a hair ball. This condition is more frequent in cattle than in human beings, and Dr. Sosman showed two specimens obtained from cattle.

Dr. Harvey Cushing gave the main address of the evening on the subject of "A Pathologically Verified Example of Pituitary Basophilism." The speaker first considered the cytology of the normal pituitary gland. In the anterior lobe we find three types of epithelial cells: namely, (1) a small chromophobe or mother cell with faintly staining cytoplasm; (2) a larger cell with cytoplasm that contains acidophilic granules; (3) a still larger cell with granules that take a basophilic stain. The acidophilic elements are known to produce the hormone of growth and it is probable though less definitely proved that the basophil elements provide the sex-maturing hormone. For each of these three elements, the pathologist recognizes an adenoma. The adenomas of acidophilic and of basophilic elements are frequently very small, and one must conceive of them as being extremely active structures in order to produce such profound effects in the body. An analogy of this is the minute adenomata of the islands of Langerhans which give rise to hypoglycemia and of the parathyroid glandules which cause v. Recklinghausen's disease of the bones.

The acidophilic adenomas cause acromegaly. The basophilic adenomas have been shown to be associated with a peculiar disorder more common in women and characterized by a rapidly acquired abdominal obesity, amenorrhea, hirsutism, plethora, purplish striae atrophicae, hypertension, glycosuria, skeletal decalcification and atherosclerosis. In its variable polyglandular phases, the disorder is probably not uncommon and is usually ascribed to an adrenal hyperplasia. To the eight verified examples of this affliction so far reported, Dr. Cushing added another thoroughly studied case.

This was a healthy, energetic, young woman who in 1919, at 20 years of age, began suddenly to gain weight, became amenorrheic, hirsute, and purple cutaneous striae began to appear on her body. Because sugar was found in her urine, she was sent to a diabetic clinic where it was recognized that hers was more than a simple case of diabetes but rather what was then called a polyglandular disorder. Under multiglandular treatment she subsequently became much improved, her hirsutism disappeared, the menses returned and the glycosuria disappeared. This remission of symptoms persisted for the next several years.

At the end of 1928, her former symptoms returned in exaggerated form and she was studied in many clinics, among them the Brigham Hospital to which she was referred in October, 1932. She had very pronounced striae, her face and extremities were plethoric, she shaved daily, her blood pressure was from 220 to 240 systolic. X-ray findings showed moderate skeletal decalcification and multiple renal calculi. Dr. Aub found that her body chemistry was essentially negative except for a low sugar tolerance and an increase in nitrogen output. Three weeks after leaving the hospital, she had an acute attack of dyspnea and succumbed in a few hours to what was supposed to be acute pulmonary edema. A necropsy revealed a normal appearing pituitary body, large adrenals, a small thyroid and cystic ovaries. The aorta and blood vessels were arteriosclerotic. The anterior-pituitary body on serial sections showed a small basophilic adenoma, slides of which were exhibited, particular attention being drawn to the invasion of the posterior lobe with basophilic cells.

Dr. Cushing next considered certain anatomical facts regarding the pituitary body. This gland is in close relationship to the hypothalamus and nuclei about the third ventricle. These nuclei send large numbers of non-medullated fibres to the pituitary which arborize and make basket networks in the neurohypophysis. The gland is therefore well innervated. There are large blood sinuses in the anterior lobe which connect through the stalk with the spaces about the nuclei surrounding the third ventricle. In newborn infants dying from asphyxia these vessels, being dilated, are particularly well shown and they have been described as constituting an hypophysioportal circulation.

Sections of the pituitary and stalk in the case described above show large veins passing up into the stalk from the anterior lobe and ending about the nuclei surrounding the third ventricle. This may be considered as one route by which the secretions of the pituitary gland can reach the tuberal region.

Experiments of Dr. Zondek have shown that in the bovine gland the most anterior and posterior sections of the gland when injected into immature mice give no response. Sections closer to the centre of the gland give a slight response; while those taken from the pars intermedia produce rapid sexual maturation in the animals. The stalk, tuber cinereum and the fluid of the third ventricle also contain this maturing substance. The posterior lobe, the stalk and the nuclei about the third ventricle contain open spaces in which secretions of the posterior pituitary body normally diffuse; and this represents the principal route by which the products of the pars intermedia reach the region of the third ventricle.

Finally the speaker considered the relationship of the adenomata of the basophilic cells in the pituitary to the other endocrine glands. He pointed out the very intricate mechanism that exists in the endocrine balance, and how pituitary disturbances may produce such changes as hyperplasia of the adrenal cortex, absence of corpora lutea in the ovary, low sugar tolerance, and low basal metabolic rate.

In conclusion, Dr. Cushing stated that the uncovering of syndromes such as this begins with their clinical description. The next step is the pathological identification of the primary lesion

which in the case of the small hyperfunctioning glandular adenomas may long be difficult to disclose. The final step is the subjection of these findings to the scrutiny of experimental physiology and pathology. In this way, and in this way only, we slowly advance toward a rational treatment.

BOSTON CITY HOSPITAL

Staff Clinical Meeting

A Staff Clinical Meeting was held at the Boston City Hospital in the Thorndike Amphitheatre, March 1, at 8:15 P.M. Dr. Soma Weiss presided.

Dr. Richard P. Stetson opened the meeting with the presentation of a patient with diabetes mellitus and a concomitant liver disorder. The patient was a twelve year old boy, who has had diabetes for four years. The condition developed in 1929 after scarlet fever, furunculosis, and erysipelas. Although he was supervised carefully with regard to diet and insulin, it was difficult to keep him sugar free. He frequently suffered from diabetic acidosis. In September, 1931, while in the hospital, the liver edge could be palpated. Several months later, in another admission for acidosis, the liver edge was three fingerbreadths below the costal margin. Three weeks later it was at the level of the umbilicus. The patient was jaundiced with a question of ascites.

On exploration the liver was markedly enlarged and smooth. Biopsy showed normal liver tissue well-filled with glycogen. The preoperative glucose and insulin were probably factors contributing to this. The blood cholesterol was 115 two weeks after operation. The patient's subsequent course has been about the same. His liver has remained enlarged, and he continues to show acetone and sugar in his urine despite treatment. There is no ascites.

According to Dr. Stetson, enlargement of the liver, while not unheard of, is comparatively rare in diabetic children. This liver is remarkable only in size and glycogen content. The liver's rôle in carbohydrate metabolism is well known. It supplies the blood with glucose and also removes it, acting as a reserve supply or depot to protect against hyper- and hypoglycemia. This patient's overnight rise of the blood sugar level is due presumably to the fact that as the blood sugar approaches a relative hypoglycemia, sugar is released by the liver. The speaker suggested that this liver might be the source of the almost constant acetone in the urine. Experimentally it has been shown that the liver may add acetone to the blood, whereas other organs remove it.

Dr. Howard Root discussed some of the aspects of the relation of the liver to diabetes. During acetonuria the urobilin excretion has been shown to be high. Best has shown that the choline portion of lecithin fed to depancreatized dogs can keep the dogs going without an insulin ration. In a short time, however, the liver is found to be filled with fat. In hemochromatosis, diabetes is known to be very resistant to insulin. Dr. Root cited a case of a patient who received 1600 units of insulin daily, to no avail, in the presence of hemochromatosis.

Dr. Francis W. Palfrey was the next speaker and spoke on "Physical Signs in Pneumonia and Empyema." In pneumonia the breathing, the characteristic "pneumococcus cough," and the bronchial whisper over the area of consolidation, are among the most reliable signs. In empyema, the "washed out, septic look" is often quite typical. Attention to the tactile sense and use of the index finger in percussion are important. In pneumonia the importance of early diagnosis, on account of the availability of serum, cannot be too strongly emphasized. The history of a "stitch" in the side, the sudden onset of fever after a chill, a high white blood count (often of value in differentiating pneumonia from influenza), and tenacious, pink, rusty, or yellow sputum often lead to the diagnosis. A friction rub may be heard over the affected area early.

The shadow by x-ray may appear 24 hours before physical signs are elicited. Dullness on percussion is not a frequent early sign, and the textbook "fine showers of crepitant râles" is seldom of use. The bronchial whispered voice is most reliable. In following a given case of pneumonia, it is to be remembered that development of body immunity is probably more important than the extent of the process. Pericarditis and effusion over the involved area are to be carefully watched for. The most important signs of the latter are a change of dullness to flatness and a decrease in tactile fremitus (when it has been obtained in the first place). The area of dullness should be outlined in relation to the spinal column. The presence of Grocco's triangle of paravertebral dullness on the side opposite an area of flatness means fluid. Dullness over the vertebrae higher than the base of the sound lung means the same thing. The dullness of consolidation ends on the diseased side two fingerbreadths from the vertebral column.

In empyema, Dr. Palfrey emphasized that the patient usually perspires, looks exhausted and febrile, and is free from dyspnea. Failure of appetite and continuing emaciation are also suggestive signs. Empyema in active pneumonia is to be tapped, but is better not drained. Interlobar empyema may give no physical signs at all, but sometimes on careful percussion there may be found a narrow band of dullness above the nipple extending out toward the shoulder. X-ray films may show an empyema where physical signs fail to reveal it, or again, the lung fields may be so opaque with consolidation in the x-ray that no empyema is visible.

Dr. James M. Faulkner told of an interesting case of acute nicotine poisoning. The patient was

a 35 year old gardener, and an habitual smoker of tobacco (6 pipefuls a day), although this had nothing to do with his illness. One day he sat on a chair with "nicofume liquid" on it. "Nicofume liquid" is a 40 per cent water solution of nicotine used in garden sprays. It soaked through his clothes. Fifteen minutes later he began to sweat, felt nauseated and faint, breathed rapidly, and soon became convulsive and unconscious. His temperature was subnormal, pulse 86, irregular and weak, and the blood pressure was 110/45. Tendon reflexes could not be obtained. The urine contained 2 per cent sugar. Caffeine sodium benzoate was administered and hot water bottles and blankets were used. On the fourth day he was discharged from the hospital, but soon went into another acute attack, due to the fact that he had put on his same clothes containing the nicotine. This case is quite typical of acute nicotine poisoning.

Dr. Faulkner investigated the possibility of poisoning through the skin experimentally in cats. The solution was poured onto the unscratched abdomen, sopped around, and covered. Ten cc. of "nicofume fluid" produced death in 25 minutes, three cc. produced death in 73-98 minutes, and two cc. was just below the fatal dose. Ten cc. of "black leaf 40" (nicotine sulphate) produced no effect.

Dr. Lawrence B. Ellis discussed "The Relative Value of Kidney Function Tests." The speaker first briefly reviewed the anatomy and physiology of the kidney and discussed the modern filtration-reabsorption theory of Cushny. According to this theory, all of the plasma constituents except proteins filter through the glomerular membrane in the same concentration in which they exist in the blood. In the tubules, (a) water is reabsorbed, (b) non-threshold substances (e.g., urea) diffuse back into the plasma in varying proportions, (c) threshold substances (e.g., sugar, chlorides) are reabsorbed.

In the clinical application of renal function tests, it is to be remembered that the earliest lesions are usually glomerular in location. Two tests are of value here, the calculation of the glomerular filtrate, and the calculation of the urea clearance. The disadvantages of these tests are obvious — an elaborate laboratory set-up is required, and the greatest accuracy is necessary.

To test tubular function, various concentration tests are done — the Mosenthal two-hour test (a ten point variation in specific gravity is normal), or other concentration-dilution tests, with their many modifications, can be performed. A repetition of urinalyses, clearance tests, and concentration tests at regular intervals will aid in determining the prognosis. The phenolsulphonphthalein excretion is not markedly altered until 50 per cent of the kidney tissue is destroyed.

Dr. Edwin A. Locke spoke on "Haverhill Fever." He told of a man, 28 years old, who was admitted to the hospital complaining of pain in the epigastrium, anorexia, and vomiting of four weeks' duration. Five days before admission he had had a series of stabbing pains in the right chest. The past history and family history were unremarkable, except that the patient has always been a moderately heavy drinker. Physical examination showed a chronic alcoholic. The heart rate was 110 and regular, and there was a loud, blowing systolic murmur heard best at the apex. The temperature was 101.8°. The diagnosis was chronic alcoholism and mild upper respiratory infection.

White blood counts varied from 6,500 to 13,000; the red blood count was 4,260,000; hemoglobin was 65 per cent; the pulse ranged from 85-88. One blood culture was negative, but later ones were positive for the "Haverhill bacillus." Death ensued 17 days after entry, generalized hemorrhagic petechiae and an enlarged spleen appearing on the day before exitus. A postmortem examination was performed, the only one that has ever been done in this disease. Mitral vegetations were cultured and a pure culture of the Haverhill bacillus obtained.

Eighty-six cases of this disease, with 45 studied, appeared in Haverhill in 1926. The cardinal features were as follows: (1) a sudden onset with chill, malaise, vomiting and headache, (2) a temperature of 103-104° for three or four days, and then a drop and secondary rise 48 hours later, (3) a blotchy, irregular, maculopapular, dull red rash, and a positive tourniquet test, and (4) various arthritic manifestations. Usually on the third day, along with the recurrence of fever, many of the joints, especially the large ones, became red, hot, tender, and swollen. The organism could be isolated in pure culture from the joint fluid. There were no fatalities, although the disease persisted for a long time in some instances.

From an epidemiological standpoint, race, age, sex, sewage, water and pests played no part. There were no contact cases. The disease was finally traced to non-pasteurized milk from a particular herd of cows, although no direct evidence (cultures) was obtained from any of the cows.

Bacteriologically, Haverhillia Multiformis is a slender, gram-negative streptobacillus. In conclusion, Dr. Locke asked Dr. Place to tell briefly of his personal experiences in the Haverhill epidemic.

MASSACHUSETTS MEMORIAL HOSPITALS

The regular monthly meeting of the Surgical Staff of the Massachusetts Memorial Hospitals was held in the Trustees' Room of the Hospital on Friday, March 10, 1933, at 12 o'clock noon.

The meeting was addressed by Dr. Lester R. Whitaker, who presented an interesting "Case of Megacolon" in a child 2½ years old.

The meeting was well attended and discussion of

Dr. Whitaker's paper by several of the members followed its presentation.

RALPH C. WIGGIN, M.D., *Secretary.*

PHI LAMBDA KAPPA

The annual medical meeting sponsored by the Phi Lambda Kappa Medical Fraternity of Boston will be held Thursday, March 23, 1933, at 8:15 P.M., at the Beth Israel Hospital Amphitheatre. The following will speak:

1. Dr. Harold Blotner: Observations on the Use of Insulin in Thin People. (Slides will be shown.)
2. Dr. M. Leopold Brodny: The Bladder in Gynecology.
3. Dr. Arnold Starr. Pathological Lesions Associated with Meckel's Diverticulum. (Slides will be shown.)

Physicians, interns and students are urged to attend.

CHARLES M. KRINSKY, *Chairman.*

SUFFOLK DISTRICT MEDICAL SOCIETY

The March meeting of the Suffolk District Medical Society will be held at the Massachusetts General Hospital in the Moseley Memorial Building on Wednesday, March 29, 1933, at 8:15 P.M.

CLINICAL PROGRAM

1. Advances in Investigation and Treatment of Certain Broncho-Pulmonary Diseases. Dr. Frederick T. Lord.
2. Acute and Chronic Empyema. Dr. Wyman Whittemore.
3. Non-Tuberculous Pulmonary Suppuration. Dr. Donald S. King.
4. Intrathoracic Tumors. Dr. Edward D. Churchill.

WILLIAM H. ROBEY, M.D., *President.*
LELAND S. MCKITTRICK, M.D., *Secretary.*

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance), Tuesday evening, March 28, at 8:15 o'clock.

PROGRAM

1. Presentation of Cases.
2. Pernicious Anemia and Related Macrocytic Anemia. By Dr. William B. Castle.
3. General Aspects of Hypochromic Anemia. By Dr. George R. Minot.
4. Hypochromic Anemia in Pregnancy and Infancy. By Dr. Maurice B. Strauss.

Medical students and physicians are cordially invited to attend.

JOHN HOMANS, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

March 23—Phi Lambda Kappa. See notice elsewhere on this page.

March 28—A Lecture by Dr. E. A. Graham. See page 607, issue of March 16.

March 24—Massachusetts General Hospital. Fracture Meeting. See page 665.

March 24—Boston City Hospital. Staff Clinical Meeting. See page 612, issue of March 16.

March 26—Harvard University. Free Public Lecture on Medical Subject. See page 665.

March 28—Harvard Medical Society. See notice elsewhere on this page.

March 30—Clinics in Medicine at Peter Bent Brigham Hospital. See page 665.

April 1 and June 13—The American Board of Obstetrics and Gynecology. See page 607, issue of March 16.

April 6—New England Heart Association. See page 571, issue of March 9.

April 12—The New England Dermatological Society. See page 612, issue of March 16.

April 12—New England Physical Therapy Society. Date changed from April 19 to April 12. Detailed notice later.

April 14—William Harvey Society. See page 765, issue of October 27.

May 8, 9, and 10—American Gynecological Society. See page 516, issue of March 2.

October 9-12—American Public Health Association. See page 403, issue of February 16.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

May 3—Wednesday, Annual Meeting. Program to be announced.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

Tentative Program

Wednesday, April 5, 1933—Essex Sanatorium, Middleton. Clinic 5 P.M. Dinner 7 P.M. Speaker: Dr. Wilson G. Smillie, Professor of Public Health Administration, School of Public Health, Harvard University. Subject: Common Colds.

Thursday, May 4, 1933—Censors' Meeting, Salem Hospital, 3:30 P.M.

Tuesday, May 9, 1933—Annual Meeting. Salem Country Club, Forest Street, Peabody. Golf in afternoon; greens fee $2.00. Dinner at 7 P.M. Speaker: Dr. George Burgess Magrath. Subject to be announced later. Ladies invited.

R. E. STONE, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meeting will be held at the Weldon in Greenfield on the second Tuesday of May.

CHARLES MOLINE, M.D., Secretary.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

May, at the Meadowbrook Club, Reading.

ALLAN R. CUNNINGHAM, M.D., Secretary.

NORFOLK DISTRICT MEDICAL SOCIETY

March 28, 1933 — See notice on page 613, issue of March 16.

May—Annual Meeting. Time, place and subject to be announced.

FRANK S. CRUICKSHANK, M.D., Secretary.
1695 Beacon Street, Brookline.

SUFFOLK DISTRICT MEDICAL SOCIETY

Wednesdays at 8:15 P.M.

March 29, 1933—Clinical Program, Massachusetts General Hospital. See notice elsewhere on this page.

April 26, 1933—Annual Meeting. Election of Officers. Scientific Program—Titles and speakers to be announced later.

The Medical Profession is cordially invited to attend these meetings.

WILLIAM H. ROBEY, M.D., President,
LELAND S. McKITTRICK, M.D., Secretary

WORCESTER DISTRICT MEDICAL SOCIETY

Wednesday, April 12—Dinner and scientific meeting at the Rutland State Hospital. Scientific program to be announced later.

Wednesday, May 10—The Annual Meeting. Place and program to be announced later.

ERWIN C. MILLER, M.D., Secretary.

BOOK REVIEWS

Theoretical Principles and Practical Applicability of Alcohol Determination in Legal Medicine. (Die theoretischen Grundlagen und die praktische Verwendbarkeit der gerichtlich-medizinischen Alkoholbestimmung.) By PROFESSOR DR. E. M. P. WIDMARK, Lund, Sweden. Berlin and Vienna: Urban and Swartzenberg. 1932.

This monograph, in the series on progress of investigation in natural sciences edited by Abderhalden, is a highly technical study of the methods of investigating the behavior of alcohol in the human organism and particularly of determining the concentration and the transformation of ethyl alcohol in the living. The author is particularly concerned with the kinetic method of determination, which he describes in detail with elaborate algebraic and geometric analysis. A brief chapter at the close is bestowed on the possibly useful applicability of this method to the determination in legal medicine of degrees of alcoholic intoxication. But owing to the temperamental differences of individuals, this seems hardly to be a method of great legal value. The treatise is well illustrated by 59 figures and has an admirable bibliography of 54 titles. It is a work of interest primarily to the biochemist.

A Convenient New Pharmacology of the Medicinal Agents in Common Use

The student of medicine will always find the large standard texts on pharmacology indispensable for critical study of the actions and uses of drugs. These texts, however, are in many instances so voluminous that they are not available for ready reference or for study in spare moments.

Heretofore there has been no comprehensive, small-sized work on pharmacology. To meet this need Dr. Stanley Coulter, Dean Emeritus of the Purdue University School of Science, spent over three years in the preparation of a compact treatise on the pharmacology of the drugs now in common use by the medical profession.

The subjects are alphabetically arranged for quick reference. Under each title there is a terse statement of the constituents of the drug, its physiological action, dosage, and brief mention of its more important therapeutic uses.

This Pharmacology is prepared with special attention to the needs of the medical student.

The book is published by Eli Lilly and Company, Indianapolis; price 50 cents per copy postpaid.

Le Traitement de la Tuberculose par l'Antigène Méthylique (Antigénothérapie). By L. NÈGRE et A. BOQUET, Chefs de Service a l'Institut Pasteur. Published by Masson et Cie. Price 35 fr.

This book is a monograph of some 200 pages with various illustrations and an elaborate bibliography giving a description of the development of what is called "l'Antigéne méthylique" at the Pasteur Institute which is nothing more or less than a form of tuberculin. By substituting methyl for ethyl alcohol, the writers believe that they have developed a tuberculin which contains certain phosphorus products of the bacilli. This is what they call "phosphaties", soluble in methyl alcohol alone and to which they give the name of "antigenes".

In the preface by Dr. Calmette he gives a general summary of this monograph. They believe that this form of tuberculin has a particularly beneficial effect in the non-pulmonary forms of the disease. Their clinical results are presented in brief along with their technique for administering this product.

This of course represents one more attempt—and a most laudable one—to find a specific against tuberculosis. It is well worthy of further trial where the results can be watched and carefully checked but certainly is not intended for general use for the present at least.

A Text-Book of Anatomy and Physiology. By JESSE FEIRING WILLIAMS, M.D. Philadelphia and London: W. B. Saunders Company. 1932. Price $2.75.

This text-book of anatomy and physiology, designed for students of nursing, physical education, physiotherapy, occupational therapy, and the household arts, differs from others of its kind chiefly in its arrangement, teaching helps, emphasis, and illustrations. It presents the basic facts of bodily structure and function logically and biologically, with stimulative questions at the close of each chapter. In this fourth edition, the chapter on physiology of digestion has been completely rewritten, important additions have been made in the physiology of circulation and the nervous system, and the sections on embryology have been extensively amplified. Many new illustrations have been added, making a total of 416 of which 32 are in color. The B. N. A. terminology has been parenthetically introduced. The book should continue its usefulness to the group of students for whom it is intended and whom it has so well served since its first publication in 1923.

An Introduction to Dermatology. By NORMAN WALKER, M.D. Assisted by G. H. PERCIVAL, M.D. Ninth Edition. William Wood & Company, New York, 1932. 372 pp. Price $7.00.

This ninth edition of Sir Norman Walker's book will be welcomed by the many familiar with his work. The delightful personality of Dr. Walker is reflected throughout the volume. It is a personal book of 372 pages, based largely on his lectures in Edinburgh, and is one of the most readable books on skin diseases which has been published. The student cannot fail to be impressed by the vivid pictures painted, and by the author's comments on the more common and less rare skin diseases. The detail of treatment is also well carried out. There are numerous excellent colored plates from wax models besides numerous other illustrations and pathological drawings.